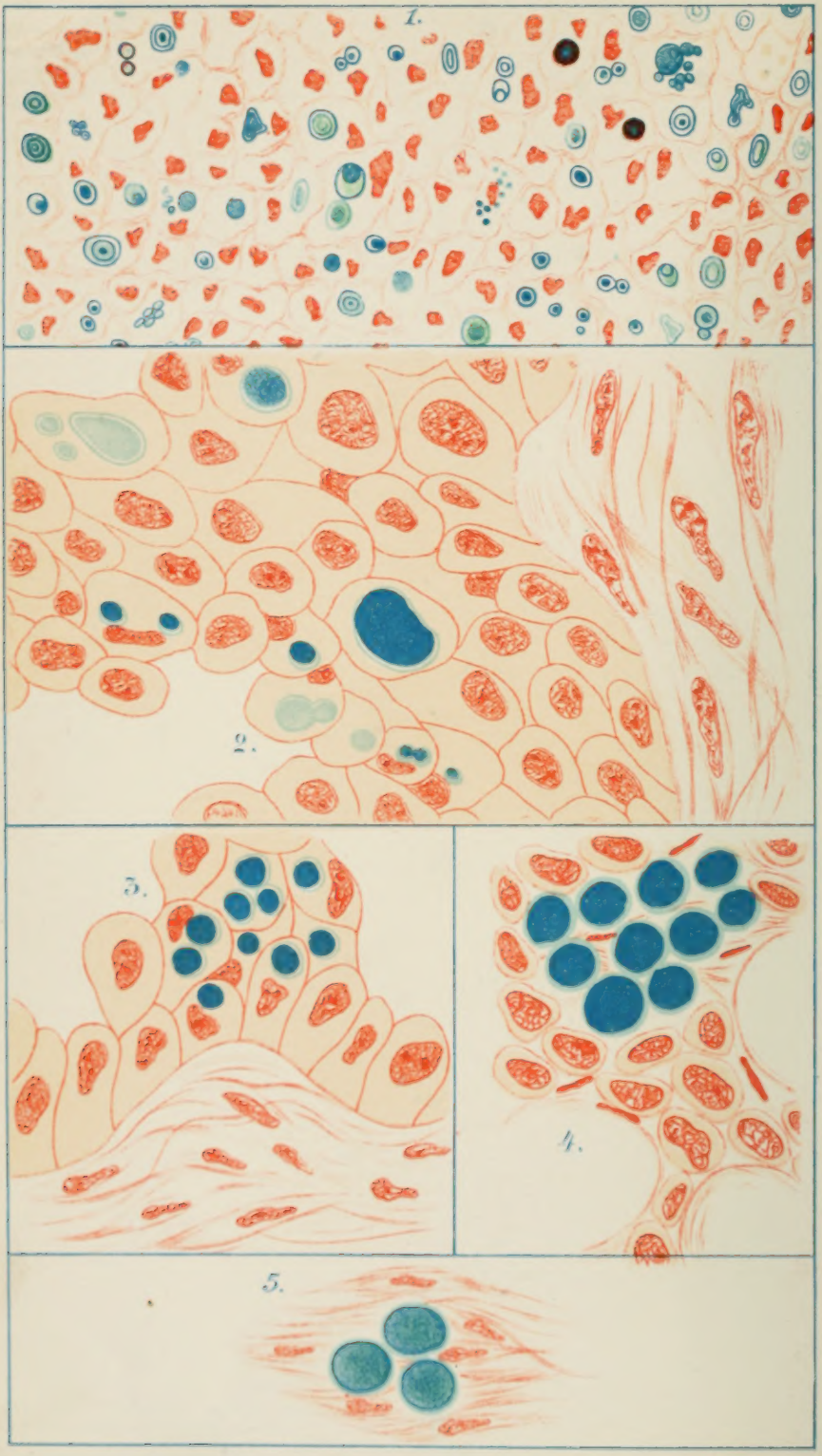
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BLASTOMYCETES IN CANCER.

Description of the plate illustrating Professor Roncali's article on "The Rôle of the Blastomycetes in Cancer." (Page 176.)

Fig. 1.—Section of an adenocarcinoma of the ovary in which are to be seen cells that resemble those of the corpus luteum and which hold in their interior a number of blastomycetes in various stages of development. One can see the very young, the young, the mature, the old, and finally the cells which have been fixed in the very act of budding. On this plate can also be seen the well-known phenomenon of metakromotosis which has been mentioned by various authors. The multiple infection of neoplasm cells described by Soudakewitch is also to be seen in this preparation. The illustration gives one a very good idea of what an extraordinary number of blastomycetes one can see in a single microscopic field.

Fig. 2.—Section of another carcinoma of the ovary in which the blastomycetes, some of them fixed in the act of reduplication, are contained in the cell-bodies of the epithelial elements.

Figs. 3 and 5.—Section through secondary metastasis of omentum which occurred in the preceding case of adenocarcinoma, and in which the parasites can be seen between the bundles of the connective and fat tissue of the omentum, many of them surrounded by leucocytes.

Fig. 4.—Section which shows blastomycetes contained in the outer layer of epithelium of a cyst of the adenocarcinoma illustrated in Fig. 2. All of the sections are stained by the ordinary mixture of safranin and malachite green.

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ON

MEDICINE, NEUROLOGY, SURGERY, THERAPEUTICS, OBSTETRICS, PÆDIATRICS, PATHOLOGY, DERMATOLOGY, DISEASES OF THE EYE, EAR, NOSE, AND THROAT, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION THROUGHOUT THE WORLD

EDITED BY

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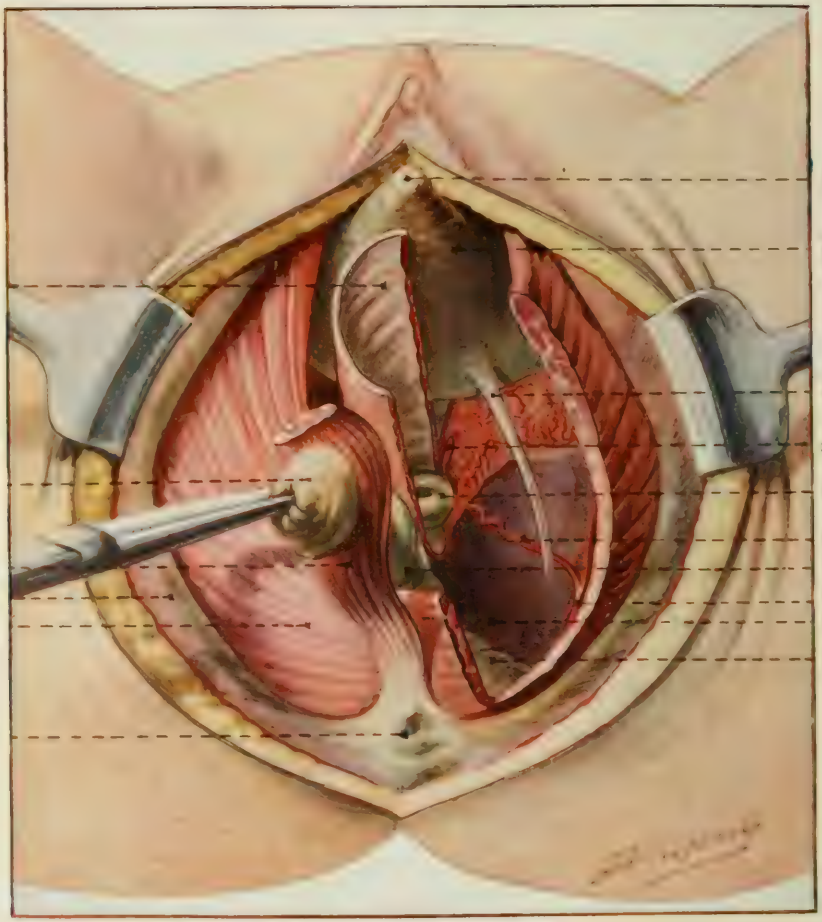

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Therapeutics

THE UNITED STATES PHARMACOPŒIA.

BY H. C. WOOD, M.D., LL.D.,
President of the Conventions of 1890 and 1900.

Among the curiosities of medical history is that of the United States Pharmacopœia. As long ago as 1778 the needs of the colonists drawn together in battle array led to the publication in the city of Philadelphia of a small military pharmacopœia, which, however, entirely disappeared from sight with the oncoming of peace after the Revolution, so that no attempt to bring order out of the chaos of drugs and their preparations was really made in the United States until 1808, when the councillors of the Massachusetts Medical Society issued a pharmacopœia to meet the local needs of New England. In 1820 were put forth the first efforts to create a really national United States Pharmacopœia, and at that time may be said to have been inaugurated the system of pharmacopœial production and revision which has lasted until the present year. In accordance with this system a pharmacopœial convention has met every ten years since. The Conventions of 1820 and 1830 were composed solely of physicians, who occupied themselves directly with the revision of the Pharmacopœia. By 1840 the system of direct revision by the Convention had become too cumbersome and expensive to be practical. The Convention, therefore, delegated the actual work of revision to a committee, contenting itself with fixing the lines along which the revision should progress. Moreover, the Convention of 1840 took the highly important and very proper step of inviting the profession of pharmacy to send delegates to the next decennial convention, so that since 1850 the Pharmacopœia of the United States has been the result of the united labors of the two professions.

A national pharmacopœia is, or ought to be, of the nature of a

law-book. It is, or should be, the legal standard in accordance with which apothecaries are to form their judgments as to the purity of crude drugs and make the preparations of these drugs for use by physicians. Very properly, therefore, in every European country it has governmental authority, and is prepared or revised in accordance with orders or methods given or provided for by the government, and its authority is enforced in the law courts. In the United States the national standard of drugs has hitherto been prepared under the auspices of a decennial voluntary convention, which, not being incorporated, has had no legal existence. Nevertheless, owing to the voluntary submission of the professions of medicine and of pharmacy to the Pharmacopœia, and owing to the extraordinary growth of the general commonwealth, the work of the Convention has grown in extent and importance until committees which have no legal standing have come to control in a measure the practice of medicine in the United States, to handle many thousands of dollars, and to be clothed with powers which might have been used for personal gain, but which have always been administered for the general good. The long continuance of the system has been largely due not only to the high character and learning of those connected with it, but also to the extraordinary popularity of some of the commentaries upon the Pharmacopœia. Without the universal acceptance of these commentaries the Pharmacopœia, which has no direct legal authority, would hardly have maintained its hold for three-quarters of a century.

The most successful of these commentaries—namely, the United States Dispensatory—was prepared and published by Dr. George B. Wood and Dr. Franklin Bache, not with any expectation of making money out of it, but for the purpose of enforcing, if possible, the dictates of the Pharmacopœia. The first United States Pharmacopœia, that of 1820, had so far failed to command respect and obedience that many of those connected with its creation were inclined to believe that it was not possible to obtain voluntary conformity to a standard which had no legal authority; but the thought came to Drs. Wood and Bache that it might be possible to establish the Pharmacopœia with the aid of a thoroughly useful commentary. The success of their conception and efforts was immediate and extraordinary.

The correlation between the Pharmacopœia and its commen-

taries has not gone unchallenged. Members of both professions have from time to time failed to grasp the true nature of a pharmacopœia as contrasted with that of a dispensatory, and some have believed that the United States Dispensatory was a monopoly feeding upon the Pharmacopœia, when, in fact, the use of the Pharmacopœia for the purposes of comment has at all times been free to all, and the market has at all times been open to any one who desired to rival existing commentaries. This is as it should be. In the matter of the Pharmacopœia the Convention practically stands in the place of the government, and the Pharmacopœia published by it, to be successful, must be as open to comment and use as is any public document put forth by the government. It is, in other words, a public book for the public weal, to be used freely, either in the study or in the drug-store, by any one to whom it may be useful.

At one time there was very serious danger that a rival pharmacopœia would be published under the auspices of the American Medical Association. Fortunately, however, for the good of the general profession and of the public, the American Medical Association, after a consideration of the subject lasting from one annual meeting to another, and after a very thorough debate, wisely decided to be satisfied with what had been done, and to make no attempt at the formation of a drug-standard distinct from and independent of that already existing; thereby preventing the appearance of two rival pharmacopœias on the American horizon, with the consequent clash among followers and the consequent confusion among apothecaries.

It is a matter of great importance that the true scope of the Pharmacopœia should be recognized by both the medical and the pharmaceutical professions. The frequently spoken of and probably wide-spread feeling, that the United States Pharmacopœia is not properly appreciated and supported by the medical profession of the United States, is apparently to some extent founded upon a misunderstanding. A pharmacopœia is not intended to be a guide to the practice of medicine or in any way a book which shall be used by the doctor in his daily life. It is not prepared for the purposes of instructing the medical profession, but of guiding pharmacists. It could be so popularized as to reach in the medical profession a considerable sale only by making it a treatise on thera-

peutics,—in other words, by causing it to cease to be a pharmacopœia. All that the doctor must know concerning the Pharmacopœia is what drugs are named by it as standard remedies, and what preparations are prepared according to its directions by the apothecaries. This much of knowledge concerning the Pharmacopœia the physician gets from his text-books and dispensatories. On the other hand, the Pharmacopœia is the *vade mecum* of the apothecary, with which in hand he does his work, and its pecuniary support must come from him. The Pharmacopœia has for its intent the furnishing of the standard by which the druggist may judge and be judged as to the character and purity of the drugs which he buys and sells, and from which he may get directions as to the methods by which the preparation of these drugs must be made. Its most important function is to make sure that, when a certain preparation—*e.g.*, a tincture—is written for by the New York physician in his office, the apothecary in New Orleans shall put up this prescription with drugs of a definite strength known to his far-off colleague. The Pharmacopœia should furnish, in clear, plain, technical language, physical and chemical tests by which the purity of drugs may be determined; it should give formulas for the making of the preparations of these drugs; and it should not give other information or attempt to be a hand-book of chemistry, of materia medica, or of therapeutics. In verity it should be a bare chart for the reading and application of whose directions previous technical training should be necessary.

As the Pharmacopœia is intended for the use of the apothecary, it is plain that it should be chiefly prepared or revised by the apothecary. It seems absurd to ask a physician to lay down the tests for, or to invent the method of making the preparations of, a certain drug. The most that the doctor has to do in a revision of the Pharmacopœia is to decide what drugs are sufficiently used or useful in the treatment of the sick to warrant their being recognized as standard remedies. Very properly, in accordance with this, the later pharmacopœial conventions have followed the custom of selecting a medical practitioner for the President of the Convention, but of making an apothecary the chairman of the committee to which the revision of the Pharmacopœia is intrusted after the great principles have been laid down by the Convention, and has further seen to it that the majority of this committee shall be drug-

gists and chemists. These things do not make the Pharmacopœia of less interest or importance to the physician; without the Pharmacopœia satisfactory medical practice would be simply impossible; but they do make the relations of the professions to the book rather indirect than direct.

Another erroneous opinion in regard to the Pharmacopœia, which has had some hold upon the medical mind in the United States, is that it should give decisions as to the value of newly offered drugs. This is plainly, however, outside of its province. Any new drug must be tested on its own merits by the profession, and should not gain entrance into the Pharmacopœial list until the profession has determined its value. In such matters the Pharmacopœia must follow, not lead, the professional judgment.

The same is true in regard to old remedies which are falling into disuse. The revisers of the Pharmacopœia can dismiss a drug only when it has fallen into desuetude. It would be intolerable to allow half a dozen doctors, however eminent, to say that a remedy in whose value they had no belief must not be employed, or to decide, on the other hand, that a certain untried candidate for medical use should be given official sanction. The revisers ought simply to ask themselves is this drug sufficiently in use to require that the apothecary shall be given official directions as to its physical and chemical character and as to the method in which its preparations shall be made? They should further remember that geographically they represent but a small portion of the United States, and that in a country so large as ours certain drugs may be employed in one section which are not used in another, and therefore should be admitted to the Pharmacopœia although perchance none of the revisers ever themselves use them. Thus, to illustrate, if there were a large demand for powdered quartz by the physicians of a certain considerable division of the country, the Pharmacopœia ought to give instructions as to determining the purity of the quartz and the best methods of making its powder, even though the revisers of the Pharmacopœia should believe the remedy to be inert.

When I came to study the history of the United States Pharmacopœia for the preparation of my Presidential Address before the Convention of the present year, it seemed to me apparent that the time had come when a legal existence should be given to the Con-

vention by incorporation. The money handled by the Committee of Revision of the Convention had, during the past decade, amounted to many thousands of dollars. The question as to the ownership of this money had been raised and very earnestly debated. It was asserted, on the one hand, that these moneys were legally the property of those individuals who had prepared the manuscript, had made the contracts and attended to the publication, and in whose names, or at least in the names of some of whom, the copyrights had been taken out. Further, as some of these members of the Committee of Revision had died, it was urged that their heirs had a legal claim upon these funds, and that members of the Committee of Revision would be personally responsible to these heirs if the money in the possession of the treasurer of the committee was handed over to the Convention of 1900. It is true that, after a declaration had been obtained from very high legal authority that these statements were contrary to the proper reading of the law, and that the money belonged in law as well as in equity to the Convention, the Committee of Revision almost unanimously agreed to the transfer of the money, which was finally effected; but it was plain that at some future time the question might be reopened and disagreeable controversies, if not serious consequences, might ensue. Moreover, it seemed to me evident that, with the growth of membership, the occasion might arise in which it would be very difficult for the President to apply parliamentary methods to an unwilling Convention which had no legal existence.

Again, in the work hitherto done by the committees of revision appointed by successive pharmacopœial conventions, two entirely distinct functions—namely, revision and publication of the Pharmacopœia—have been carried out. Revision is a matter of applied science; publication and distribution is a matter of business; so that for the performance of the two functions of the committee there are required different qualities of mind and of training, which may, but usually do not, coexist in a high degree in one person. Further, it is but just that chemists or other specialists who do the scientific and literary work of the revision of the Pharmacopœia should receive compensation for their labors if funds be available. It is intolerable to ask a gentleman working as an expert to fix his own remuneration from public moneys contributed for the general good. Plainly the Convention should have two active Executive

Committees, one composed of practical scientists and one of business men. These and similar considerations led, after consultation with Professors Rice, Remington, and others, to the proposal, in my Annual Address, of the incorporation of the Pharmacopœial Convention, with a Constitution and By-Laws that should require the creation of a Board of Trustees which should attend to the business interests of the Pharmacopœia, and of a Committee of Revision which should have charge of the preparation and printing of the manuscript. After much discussion the plan was in its general scope approved by the Convention, a Constitution and By-Laws were adopted, and instructions were given to those selected as the Board of Trustees to proceed to incorporation under the laws of the District of Columbia.

Accordingly, on July 11, 1900, all legal formalities having been complied with, the United States Pharmacopœial Convention was incorporated in the District of Columbia, with a Board of Trustees composed of seven persons, the same as those who had been selected at the meeting of the Convention to constitute its Board of Trustees. Of course, the Constitution passed by the Convention was without legal binding force upon the new body, but on October 31, 1900, the seven incorporators and the seven members of the Board of Trustees named in the Certificate of Incorporation adopted a Constitution and By-Laws which, with certain modifications in verbiage made necessary by legal requirements, are the same as those adopted by the Convention of 1900.

Under the now existing conditions the Convention of the United States Pharmacopœia is a legal body, which meets every ten years, and is represented between its times of meeting by two executive committees, clothed with power to act under instructions given them by the Convention. The Constitution requires that the Convention shall be composed of delegates from incorporated medical colleges, and medical schools connected with incorporated colleges and universities; incorporated colleges of pharmacy, and pharmaceutical schools connected with incorporated universities; incorporated State medical associations; incorporated State pharmaceutical associations; the American Medical Association, the American Pharmaceutical Association, and the American Chemical Society; also, from the United States Army, the United States Navy, the Marine Hospital Service, and from all organizations whose representatives were

admitted to the Convention of 1900. The Constitution further provides for the election of a nominating committee, a committee of revision, and a board of trustees; whilst the By-Laws define the duties of these respective bodies. The especial duty of the Board of Trustees is to attend to the business matters of the Pharmacopœia, all moneys passing through their hands and all contracts being made by them; while the scientific and literary work of the Convention, up to the final preparation of the manuscript of the forthcoming Pharmacopœia and the reading of the final proof-sheets, is intrusted to the Committee of Revision. In order that the two Committees shall work together, the chairman of the Committee of Revision is *ex officio* a member of the Board of Trustees.

Thus, at the opening of the new century the United States Pharmacopœia enters upon new conditions, which can hardly fail more and more firmly to establish its authority until it shall have received that open and complete recognition by law which was hardly possible so long as it emanated from a Convention which had no legal responsibility and was therefore under no legal control.

ON THE INDICATIONS AND CONTRAINDICATIONS FOR THE USE OF DIGITALIS IN TREATING HEART DISEASE.

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL.

BY A. POTAIN, M.D.,

Professor of Clinical Medicine at the Paris Faculty.

GENTLEMEN,—The theory as to the way in which digitalis acts cannot be relied on as the only source of indications for the administration of this drug in practice. Its action in man and in animals may differ; with our patients it may differ in a healthy man and a sick one, and even with a single patient, according to what the disorder may be. Observation alone, therefore, is able to supply positive indications. However, general ideas of pathological physiology and of pharmacodynamics are very useful in interpreting the results obtained, and in guiding physicians in any new experiments made in applying this remedy.

The fact is well known that experimental research, as well as long experience, shows that digitalis slows, regularizes, and equalizes the heart's movements; it also increases systolic energy and the tonic resistance of the myocardium, counteracts peripheral stagnation, and brings about absorption of œdema. It is therefore indicated whenever the heart-beats are very quick, irregular, or unequal; when the heart is dilated from inadequate systolic contraction or tonic resistance of its walls; and when there is œdema from stasis, and urinary insufficiency through tissue infiltration and a renal circulatory stasis.

You of course understand that in heart disease digitalis can have no other effect than to moderate or remove functional disorders resulting from the heart lesion, and that it can have no effect on the lesion itself. There is therefore no reason for giving it except when these functional disturbances occur; and there can be no purpose in giving it so long as the lesion is sufficiently com-

pensated,—that is to say, so long as the circulatory obstacle is properly overcome by increased heart effort, peripheral and pulmonary circulation goes on in a normal way, and various viscera defend themselves sufficiently against the effect of the circulatory disturbance of which they are the seat.

For these reasons digitalis has little effect, or even no effect at all, when the functional disorders are due to any acute complaint of the peri-, myo-, or endocardium, whether the complaint be primitive or secondary. On the other hand, it succeeds admirably in cases of organic lesion which require from the heart an increase in energy above that which it is capable of supplying.

Among those organic lesions benefited by digitalis, mitral incompetence is the one in which the indications for the employment of this drug occur the most frequently. The greatest chance of success is offered in those cases where the drug is administered at a period of time near the origin of the disorder. This lesion is one in which compensation is difficult to establish. You understand what is meant by the term compensation in disease. It is the result of the increase in power that a muscle acquires when steadily called on to a greater effort and when its nutrition is capable of being raised to the required level. It is in this way that hypertrophy of the left ventricle is established in stenosis of the aortic orifice, and that the biceps increases in size with gymnasts and the muscles of the calf in ballet dancers.

In mitral incompetence there is nothing to induce increased function and compensatory hypertrophy of the left ventricle, so that this hypertrophy is generally lacking. On the other hand, this is a lesion that causes great fatigue of the ventricle; resistance is lessened, owing to the regurgitation of a part of the blood back into the auricle during the contraction; there is increased diastolic distention caused by the return to the ventricle of the volume of blood backed out of it during systolic contraction, and the flow of blood to the heart is rendered unequal by respiratory influences. Under such conditions digitalis increases systolic energy, through which a larger quantity of blood is sent into the aorta, while at the same time it increases the tonic resistance of the walls of the heart and lessens irregularity due to their at times excessive distention. To this can be added the fact that it stimulates the tonicity of the capillaries and thus regulates peripheral circula-

tion; it is likewise probable that it stimulates the pulmonary capillaries, and protects to a certain degree the menaced statics of the right side of the heart. For these reasons digitalis produces in this class of cases results that are in many instances wonderful.

The situation is, however, not quite the same in mitral stenosis. This lesion is well tolerated for long periods; the obstacle to the circulation is fixed and constant and does not give rise to cardiac irregularities; it only imposes on the left auricle a supplementary effort which causes the walls to hypertrophy in a regular manner. Simple rest, which lessens the work thrown on the heart, is sufficient to put an end to the symptoms for a long period. Still, the time does come when pulmonary stasis and the dilatation of the right cavities resulting from it bring on signs of asystoly. At that moment the use of digitalis may be advantageous in various ways. It lengthens the diastolic pause during which the blood passes from auricle to ventricle; it stimulates the wavering energy of the left auricle and right ventricle; it lessens the peripheral circulation and thereby the volume of blood reaching the heart through the narrowed orifice; finally, it is possible that it increases the tonus of the pulmonary vessels and lessens stasis in the lungs.

Spontaneous compensation also occurs in aortic stenosis, and the ventricular hypertrophy increases in equal proportion to the resistance. No inequality of rhythm nor pulmonary stasis are observed in this form of heart disorder. When for any reason the muscle weakens, asystoly may take place, in which case digitalis will be of service; its indication is, however, very unusual, and occurs only at a late period in the evolution of the cardiac disorder.

This indication is also rare in aortic incompetence; in fact, some writers have asserted that in this form the use of digitalis can only be harmful, as it slows the heart's beats and lengthens the diastolic period, which, in this form, is the unfavorable one. It is evident that, were this period to be so lengthened that all the blood propelled during systolic contraction had time to fall back into the ventricular cavity during the diastolic period, there would be no circulation at all. Nevertheless, digitalis may be of some service to patients with tachycardia, as too rapid heart action in aortic incompetence is not without drawbacks. The beginning of diastole is the moment when the greatest aortic pressure occurs, as well as the maximum of regurgitation into the ventricle; then

the flow decreases progressively, as is shown by the lessening in the murmur sound. Too rapid action, therefore, reduces diastole to the period during which the maximum of regurgitation occurs, and it is then that digitalis may intervene efficaciously by bringing back the heart-beats to their average frequency.

Digitalis must not be used in the therapeusis of interventricular perforation, in which disorder its administration would be absolutely useless, as this lesion is always wonderfully well tolerated.

Tricuspid incompetence is not in itself an indication for the use of digitalis; but this disturbance is rarely primitive, and is almost always connected with other cardiac lesions which require digitalis. This drug is capable of rapidly and completely putting a stop to incompetence, but its use is not always free from drawbacks, as I shall show you in a moment.

Cardiac hypertrophy is seldom found existing by itself, though this is sometimes the case in neuritis of the left brachial plexus. It is then followed by none of the accidents that call for the use of digitalis; this drug has no action on the mere hypertrophy, which it would be more apt to increase. This is equally true of the hypertrophy of atheromatous patients, in which digitalis should be used only when the myocardium weakens; in such instances it should be given with great prudence and should be closely watched, especially with old people, for fear that the myocardium should react to it to a lesser degree than the peripheral resistance.

Hypertrophy in Bright's disease does not call for digitalis until a late stage in the disorder, as it is accompanied neither by tachycardia, irregular movements, nor noticeable œdema. Some writers have even said that arterial hypertension is a contraindication to its use; but the truth is that it only raises the pressure very slightly compared with the great increase of pressure observed with these patients, and it is capable of being of service in the last phases of the disease, when anasarca and cardiac weakness appear, or when the hypertrophy becomes complicated with some orificial lesion.

We have also to consider the various forms of dilated heart,—that is to say, the cases in which the enlargement of the cavities is more important than the hypertrophy of its walls. When there is general dilatation, accompanied by acute pericarditis, or ulcerative endocarditis, digitalis may be indicated, but it will then succeed only to a moderate degree. This is also true of dilatation due to

insufficient nutrition of the myocardium. When dilatation affects only the cavities of the right side, physicians do not agree as to the use of digitalis. According to Hoffmann, of Dorpat, it is then of no use; Penzoldt, of Vienna, on the contrary, thinks it is frequently useful; whereas Leyden asserts that it acts only when this condition depends on mitral incompetence. I think that dilatation dependent on mitral lesions can be benefited by the use of digitalis, which, on the other hand, is of little service in dilatation caused by emphysema or pulmonary sclerosis. It is often harmful in cases of dilatation brought on by gastric or intestinal dyspepsia, because it then helps to increase the dyspeptic symptoms.

Digitalis has likewise little action in functional disturbances of the heart. It has little or no control over tachycardia, whether nervous or symptomatic of Graves's disease or aortitis. Its influence on palpitations varies extremely, according to their nature. Although it sometimes relieves those that are due to simple irregular action of the heart, it usually increases such as depend on gastric disturbance. Finally, it has no action on hysterical palpitations connected with hyperæsthesia of the precordial wall, which, on the other hand, are rapidly relieved by the application of a blister.

From all that precedes you will see that the indications for the use of digitalis are, after all, not very numerous. They are: 1. Tachycardia and irregular and unequal heart action. 2. Inadequate contractile or tonic energy of the myocardium, anasarca, and peripheral stasis.

We must now consider the second half of our subject,—the contraindications to the use of digitalis.

I have already told you that among these you must not place a slow pulse. Digitalis may even quicken the number of beats in certain forms of permanent slow pulse, when the low rhythm is apparent only and depends on unequal pulsation. We have also seen that the arterial hypertension of atheromatous and Bright's disease patients is not in itself an obstacle to the use of digitalis, the increased tension it produces being insignificant relatively to the tension of nephritic patients.

A more serious contraindication is found in degeneration of the heart muscle. In such cases, if the cardiac energy is no longer capable of stimulation, digitalis becomes dangerous in that it in-

creases the peripheral resistance without increasing in equal measure the vigor of the heart itself. For this reason some writers (Leyden, Penzoldt, and Patton) look on changes in the muscle of the heart as an absolute contraindication. Others (Wood and Handfield) are less positive. The fact is that cardiac degeneration is always difficult to detect, and the clinical determination of the degree and extent of this condition is a delicate matter. It is therefore wise to be prudent in giving digitalis to old people and to those in whom you have reason to doubt the integrity of the tissue of the myocardium.

In 1888 Morse asserted that digitalis can do harm to cases of aortic incompetence only; but I have already told you, and this is also Balfour's opinion (1891), that excessive tachycardia has very undesirable results with these patients, because it keeps arterial pressure at a point at which regurgitation is at its maximum. Digitalis may then be of real service.

Although tricuspid incompetence is one of the chief indications of this drug, since no other cardiac disorder disappears so rapidly and completely under its influence, you doubtless know that its administration in these cases requires great tact, as the treatment is capable of giving rise to serious complications which would not have occurred without the absorption of the drug. The too rapid disappearance of a functional incompetence causes a sudden elevation of pressure in the pulmonary vessels; the lung capillaries, suddenly brought under the influence of a pressure to which they have lost the habit, allow themselves to be distended to the point of breaking. To avoid this sort of pulmonary apoplexy I use, in these cases, only small doses of digitalis,—that is to say, instead of giving one milligramme of digitalin in a single dose, I spread this amount over a period of five days, giving only a fifth of a milligramme *per diem*. In spite of this measure of precaution, it may be well to remind you that a case of this form of pulmonary apoplexy occurred in our wards quite recently after giving to a male patient one fifth of a milligramme of digitalin for only four consecutive days. The man was, however, a consumptive also, with cavities in his lungs, as well as a sufferer from sclerosis of the heart muscle, and his pulmonary vessels were undoubtedly very fragile. However this may be, the accident in question was not the cause of his death.

Dyspeptic disturbances may also be a cause of contraindications; such disorders are frequently aggravated by giving digitalis, particularly in the form of powder or of infusion of the leaves, and they react most disadvantageously on the heart. Another reason for refraining from giving digitalis is to be found in a cachectic condition of the patient, whether this be senile or anæmic cachexia. A final contraindication is the use of the drug itself. Repeated doses accumulate their effect and produce digitalis saturation, and in this way little by little you reach the period of accidents, of which I wish to say a few words in closing.

We need not consider acute intoxication by digitalis, as it never occurs with the doses used in therapeutics. But therapeutic doses can give rise to accidents with which you are not very familiar, as physicians are rather loath to publish them. Duroziez collected about twenty such cases, and I also have seen several.

This first thing to occur is a slight degree of nocturnal delirium, which often escapes notice when the patients are not closely watched. Next comes prostration, pallor, and cold hands and feet; finally, agitation and great distress. The pallor increases, the pupils narrow, syncope is imminent, and the patient may die suddenly. In other cases they fall into a comatose condition.

These serious symptoms occur chiefly in cases of Bright's disease, cirrhosis, anæmia, aortic incompetence, or when large doses of digitalis are given during typhoid fever or delirium tremens. They often follow the prolonged administrations of little doses which accumulate their effect, or of a single large dose in a patient already under digitalis treatment. In some instances the accidents occur in a manner entirely unforeseen, having been preceded by neither nausea nor a slow pulse.

I saw a case of this kind in our wards,—a patient who, without my knowledge, had continued to take digitalis pills for a long time after I had directed him to stop. This man grew pale and had a little nocturnal delirium; his eyes were haggard and the pupils narrowed. But the symptoms disappeared when the pills were stopped. You probably remember having seen quite recently in our wards a woman grow pale and complain of distress and slight delirium. She was suffering from extensive aortitis, and we had been giving her digitalis in slight doses. She did not die from digitalis intoxication, as she had neither contracted pupils nor ex-

cessive slowness of pulse, and *post mortem* revealed neither tetanic contraction of the heart nor cerebral anæmia. It is likely, however, that the digitalis took a certain part in causing the symptoms manifested by this patient.

The accidents due to digitalis appear in some instances to be caused by ischæmia of the cerebro-spinal system; but they may occur without any noticeable peripheral ischæmia, and should then be attributed to direct action of the poison on the nervous centres. In 1895 an account of five cases of acute psychosis, due to the administration of from two to four doses of digitalis, was published by one writer. The mental derangement consisted mainly in melancholic delirium with ideas of suicide and persecution.

On the whole, digitalis is a very useful remedy and one that cannot be replaced by any other known drug. But it must be used with much discretion, because in time its effect becomes lost, and because it may give rise to serious accidents, either when given in too large a dose or when the proper dose is continued for too long a period.

MASSAGE IN RAYNAUD'S DISEASE (SYMMETRICAL GANGRENE).

READ BEFORE THE NORFOLK (MASSACHUSETTS) DISTRICT MEDICAL SOCIETY.

BY DOUGLAS GRAHAM, M.D.,

Of Boston, Massachusetts.

Early in March, 1899, I perused an interesting "Report on Massage at the Surgical Clinic of the University of Berlin," by Professor Zabludowski.[1] I confess, however, that my faith was severely taxed when I read that, in a case of weakness of the heart following influenza, one leg which had not been *masséed* became gangrenous and had to be amputated, and that the other leg soon after began to present similar appearances, but became normal under careful massage. While I was speculating on whether this could possibly be true, an elderly lady called at my office, and told me that ten months previously her husband's right leg had to be amputated on account of gangrene; that the left one seemed to be similarly affected, and that her physician, Dr. Samuel Crowell, had sent her to me to learn if massage would be of any use. (A few days before, a very thoughtful patient, in no way connected with this case, remarked to me that massage ought to be good for gangrene; so this matter seemed to be in the air.)

No time was lost in getting to such an interesting patient. I found him to be an unusually intelligent man, a baker by occupation, sixty years of age, and fairly well nourished, considering that he had suffered from saccharine diabetes for fourteen years, for which he was under suitable diet and medication by Dr. Crowell, and doing well in this respect. For fifteen months the left leg had presented appearances similar to those that led to amputation of the right one. I found it pale, cold, and bloodless, although it was warmly wrapped. *The pulse could not be felt in the dorsal artery of the foot nor in the posterior tibial.* There was a dark

[1] Sammlung klinischer Vorträge, No. 209.

scab on the outside of the metatarso-phalangeal joint of the little toe, one at the end of the great toe, and one on the inside of the ball of the great toe. The last was one inch long, half an inch wide, and seemed to extend rather deeply, though it was but three weeks old. He had had ten of these black scabs come and go within the preceding year. Some of those on the outer aspect of the foot had been comparatively large. For a long time before the appearance of any dark spots, he had suffered severely from pain, numbness, and other disagreeable feelings in both feet by day and night. During the previous six months the foot had been growing steadily worse, and the question of amputation was being considered. His pulse was 120, heart-sounds normal, and he was of a cheerful disposition notwithstanding the gloomy prospect.

After half an hour's massage, possibly less, the whole leg and foot were warmer, the tissues suppler, and tension less, and *the dorsal artery of the foot could be felt well enough to count the pulse.* To the patient the leg felt in a glow, and the pulse was 102. My delight and astonishment were almost unbounded at seeing a dead leg brought back to life in such a short time. I could hardly believe the evidence of my own senses. Directions were left to bathe the leg well in warm water, and to rub it every morning with warm olive oil. Next day I learned that after my visit the previous afternoon the leg had continued to feel warm the rest of that day and all night, and I could still count the pulse in the dorsalis pedis artery before massage.

The method of working was principally by deep manipulation in a downward direction, to aid the arterial current; followed and alternated by upward friction, or *effleurage,* to aid the venous and lymphatic flow. To this were added, at the second and subsequent visits, resistive movements of flexion and extension of the foot and leg. After these procedures at the second visit, the pulse in the foot was more perceptible than before.

The patient had been confined to the house and the same floor for fourteen months, much of this time to the same room, and only allowed to go from his bed to his chair by the window on crutches, —a few steps. I told him to walk into his front parlor on the same floor once an hour, and to have his body rolled once daily with a baker's roller by one of his own family. His general strength rapidly increased on this plan, and he was allowed gradually to

return to a mixed diet, and did well on this.[1] In a few days he was walking twice every half-hour to the front room and back again to his bedroom. At the end of a week it was evident that the large dark eschar on the inside of the ball of the great toe was growing pale in the centre, and that its margins were diminishing by the encroachment of healthy skin. Five days later this improvement was still more marked, and the patient was very cheerful and hopeful. I visited him every day for a time, and thereafter every other day, getting valuable assistance from his wife, who was a most faithful nurse. He was able to go down-stairs and enjoy life in five weeks from the time I first saw him.

The progress of this case, however, was not without anxiety or interruption. About four days later there came a light purplish appearance around the scab at the end of the great toe, which was temporarily lessened by the derivating effect of massage around it. Four days after this the purplish region had increased, so that it covered the whole of the top of the great toe and also the inner aspect as far as the terminal articulation. It had become darker in color, somewhat like a purple grape, and had a blistered appearance, as if it had been scalded. To the touch it did not seem warmer than the surrounding skin; to the patient it felt very uncomfortable. He had had such symptoms before, and they had been premonitory of something worse. His exercise of walking twice every hour to the front room and back was decreased to once an hour, the temperature of the water with which the leg was bathed was reduced, and massage was resumed oftener, for we had been gradually slacking up on this. In a week there was decided improvement, and he was again going down-stairs once a day. The dark spots with purple margins gradually faded away and disappeared, and he was doing so well that he was left to the care of his wife. The whole period of my attendance was a little less than two months. Then I no longer doubted the statement of Zabludowski,—that a leg presenting the appearance incipient of gangrene had been saved by massage and remedial movements.

A year later I called to see how this patient was getting along,

[1] Since this article was written, I have learned from Dr. Crowell that this patient's urine always contained sugar in about the same quantity whether he was on a diabetic diet or not, and during the last five years of his life albumen also was present.

and found the foot and leg looking quite natural, no dark spots nor unnatural color anywhere, and he was wearing an ordinary stocking. The pulse could be felt in the dorsal artery of the foot as well as in the posterior tibial; and after fifteen minutes of massage by way of experiment it became fuller and stronger and the veins more clearly visible. He had had the grippe, the preceding winter, which left him worse than it found him; and within the previous month he had had two severe attacks of dyspnœa with great distress in the region of the heart, notwithstanding which he had still persisted in going up and down stairs. His appetite was failing; he no longer cared for his pipe, which till a short time before had been an unfailing source of comfort; he looked pale and thin, and his eyesight had become affected so that he could not read nor see at a distance, and something obscured his vision, though he could distinguish faces and large objects. It was evident that he was not long for this world. His disturbance of vision I naturally supposed must be due to diabetic, or possibly albuminuric, retinitis.[1] But fancy my surprise when I called a month later and learned that his vision had returned, so that he could see to read perfectly, after his visual trouble had lasted for two months. So we must regard the ocular episode as due to spasm of the central artery of the retina and its branches, similar to what occurs in other arteries in this curious affection.

Without suspecting its nature, Loring, in his "Text-Book of Ophthalmology," 1891, well describes the appearances, under the title of ischæmia of the retina, in which the arteries are reduced in size or are even thread-like, while the veins remain of normal dimensions or are fuller than usual. He was looking right at Raynaud's disease and did not know it.

Professor J. Collins Warren[2] was so fortunate on one occasion as to have ocular demonstration of this arterial spasm in a case in which temporary disturbance of vision occurred; the ophthalmoscope showed a well-marked contraction of the central artery

[1] Since the above was written, I have learned that he was seen at this time by an oculist, whose report confirmed my suspicions, inasmuch as he found on ophthalmoscopic examination that the arteries in both eyes were diminished in size, the veins congested and tortuous, besides other appearances that he thought were due to diabetic albuminuria. The lenses were clear.

[2] Boston Medical and Surgical Journal, January 16, 1879.

of the retina. This symptom occurs sufficiently often in Raynaud's disease to make it clearly recognized. Hofmeister, of Carlsbad,[1] observed gangrene and loss of fingers in a case of diabetes.

Quite a number of years ago a patient came to me with symptoms which I did not then know how to interpret. If I had ever heard of Raynaud's disease at that time, I had certainly forgotten it. To my surprise, therefore, I find I have written my diagnosis, —*vaso-motor spasm.* Later, with a greater complexity of symptoms, an excellent consultant labelled them rheumatism. This patient was about thirty years of age, and had had the grippe eight months before, which confined her to bed four days with high fever. At first she had severe pain in the forehead, then in the back, and finally it settled in the right hand and arm, where it stayed more or less continuously until she came to me. It lasted for a few hours to two weeks at a time, but the hand and arm were never quite free from it. It was worst opposite the palmar aspect of the middle phalanges, next in the same region of the first and third phalanges, and even when comparatively free from pain, pressure here, as well as in the palm of the hand, caused pain which lasted for a few minutes. The pain extended up the arm to the shoulder. She was an accomplished pianist, and could easily play for several hours when well, but at this time she could only play for half an hour at her best, at the end of which time she had to stop from pain and weakness, and then the tips of the fingers also hurt upon pressure. *When she put the hand into cold water it turned greenish-white and felt numb.* She had had the pain steadily for two weeks when she came to me, and the pulse was small and hard. Massage at 2 P.M. made the hand and arm feel comfortable at the time and all the afternoon; but she had more pain than usual next morning, which quickly disappeared of its own accord. Again at 2 P.M. she had massage, which was accompanied and followed by increased comfort and fuller and stronger pulse.

Three weeks later I found my notes saying, that under massage three or four times a week she has steadily improved, so that when pain returns it lasts for a shorter time than before, and there is much more comfort in the intervals. At times she has the same

[1] Wien. med. Wochenschrift, No. 5, 1898.

sort of pain in the head, which is also much relieved by massage. She finds that this treatment puts the hand and arm in good condition for playing the piano. Five days later she played the piano for two and a half hours without fatigue or discomfort. But the progress of the case from this time was not so uniformly favorable as it had been; for twelve days later, coincident with the disappearance of the symptoms from the upper extremity, there came pain, stiffness, and swelling of the right knee.[1] Under massage and sodium salicylate these symptoms left the knee in about ten days, and the hand and arm resumed their former condition of pain, weakness, and numbness, and turning greenish-white when exposed to cold or put into cold water, for which the massage proved as efficient and agreeable a remedy as before.

The subsequent history of this case is long and wearisome. Suffice it to say that she had numerous and varying neuro-muscular pains, sometimes affecting the whole right side in a very severe manner, and requiring large doses of analgesics for their relief; and in the intervals tonics, alteratives, and anti-rheumatics were used. The symptoms in the hand and arm already mentioned were persistent, but were always ameliorated by massage, sometimes with the addition of the faradic current. It was five months and a half from the time I first saw her before she could be considered at all well. Evidently this case did not go beyond the first stage of Raynaud's disease,—spasm of the arteries. The other cardinal symptoms, local asphyxia and gangrene, were not present. Possibly the massage prevented their development.

There may be some doubt as to Raynaud's disease manifesting itself in a unilateral form. Dr. Solis-Cohen says he has seen one case, and others are on record.[2] Dr. E. W. Clarke, in the *Quarterly Medical Journal* of Sheffield, for July, 1897, has reported a case of Raynaud's disease in which the local stasis suddenly developed, having been preceded and accompanied by severe neuralgic pains. The fingers were first affected; then the corresponding digits of the lower extremities, together with the tip of the nose and the lobes of the ear. This case did not go on to the third stage, that

[1] Osler, in Pepper's Text-Book of Theory and Practice, refers to a case of Raynaud's disease that behaved in a similar manner to this.

[2] INTERNATIONAL CLINICS, vol. iii., 1895.

of gangrene, though retrogression of the malady was attended by exfoliation of the superficial layers of the skin of the affected parts. The result was complete and permanent cure; the treatment was massage and the faradic current (both locally and generally). At times the pain had been so great that morphine had to be used.

On theoretical grounds we would suppose that the galvanic current would give better results than the faradic. Galvanization of the spinal cord is said to modify favorably the arterial spasm, and this was the method Raynaud himself employed.

Dr. P. Kovacs, of Berlin, has narrated a case of Raynaud's disease in the person of a young woman, twenty-one years of age, a dish-washer by occupation, who was hysterical. She presented the three main symptoms of this malady—local syncope, local asphyxia, and gangrene of her fingers—for four months. When she put her hands into cold water they turned white, later becoming blue; when she put them into hot water they turned red, and later became blue. The treatment was massage and electricity, with the wearing of gloves for warmth, and tonics internally. Almost immediate good results were obtained, and in three weeks' time both hands resumed their natural appearance, and she was advised to engage in some other occupation.[1]

I fear that there are very few physicians who know who Raynaud was when he first described this interesting affection now called by his name. And until quite recently I myself was among the majority. Maurice Raynaud was a medical student in Paris when he wrote his graduating thesis, in 1862, on this peculiar malady,—dry, symmetrical gangrene. Since then numerous articles of more or less value have been contributed which only serve to confirm the clinical picture and its variations as he first described them. His theory is that this disease is a neurosis characterized by great exaggeration of the excito-motor energy of the parts of the spinal cord that control vaso-motor innervation,—the posterior and lateral gray substance, according to Oppenheim. The disease in its acute form presents three marked phases,—local syncope, local asphyxia or stagnation, and gangrene. In its chronic form there is a repetition of these. It usually attacks symmetrical parts of the human body,—the fingers and toes, sometimes the nose and ears, less fre-

[1] International Clinics, vol. ii., 1899.

quently the buttocks and other places. The local syncope, the pallor, the coldness, are explained by spasm of the arterioles. But it is not so easy to dismiss the asphyxia by saying that it is the result of complete vaso-motor paralysis. The stagnation may be due to a venous cramp, for the veins, like the arteries, are possessed of a muscular coat which is not only regulated in tone from the vaso-motor centre, but can also be increased by reflex irritation from the periphery.[1] The studies of Dr. Charles G. Cumston[2] led him to believe that it is not necessary for the calibre of the veins to be completely obstructed in order to produce gangrene when the nutrition and vitality of the tissues have been previously lowered by constriction of the arteries.

In explanation of these phenomena, the theory that neurons act by contiguity and not by continuity would fit admirably by assuming that in the first stage, that of syncope, the projections of the neurons that preside over vaso-motor constriction have become so tightly interlocked that they cannot let go until they become completely fatigued, when they relax, lose their power of contact and simultaneously their inhibitory or restraining influence over the arterioles, thus inducing the second stage, that of asphyxia or dilatation. This theory would be consistent with the generally accepted view that dry symmetrical gangrene may arise from either central or peripheral irritation, or both, the central or peripheral ganglia or neurons acting and reacting upon each other.

In support of the central origin, symptoms of Raynaud's disease have been found in affections of the brain and spinal cord. It is said to be very frequent in the stupor which sometimes succeeds acute mania or acute confusional insanity, in paretic dementia, in melancholia, and in other affections of the brain.[3] It has frequently been found in cases of locomotor ataxia and syringomyelia, and it occasionally appears along with hysteria and epilepsy.

The peripheral origin of Raynaud's disease is inferred when it is preceded by pain, numbness, and disturbed sensations. Endoarteritis and endophlebitis are considered as secondary lesions.

[1] Zerbes, Wien. med. Wochenschrift, 1898.
[2] Buffalo Medical Journal, 1898.
[3] Dr. James G. Kiernan, Medicine, Detroit, 1898.

Though the first or second stages may appear independently of each other, often the patient has no knowledge of the malady until gangrene sets in. Hochenegg found an etiological relationship between this disease and long-continued bodily straining without sufficient pause for rest; with chlorosis in young people; and with disturbances of nutrition in consequence of acute or chronic inflammatory affections and febrile infectious diseases, such as typhus, scarlatina, measles, variola, pneumonia, intermittent fever, tuberculosis, etc.[1] Colicky pains, intermittent hemoglobinuria and angioneurotic œdema are frequent in Raynaud's disease, according to some observers.

Dr. Coleman found broken-down corpuscles in the blood of the affected fingers in a case of Raynaud's disease, but not in the blood of the others.[2] It is not yet clear how neuritis may bring on gangrene, if it ever does. Besides the *rôle* the blood-vessels play, there must be an indirect influence of the nerves upon the cells, causing a trophic disturbance.

As precursors of this interesting affection, Dr. Adolf Havas mentions that it may be announced by either general or local symptoms; such as psychical alterations, depression, and feeling of anxiety; disturbances of digestion, anorexia, and vomiting; disturbances of sight and hearing, or other special sense. Locally there may be formication and furry feelings of the skin, painful pressure and feeling of stretching, hyperæsthesia, paræsthesia, and anæsthesia.[3] But as these symptoms are not only found in other maladies, but also may be entirely absent, it is evident that further developments must be awaited.

Life is not directly threatened by Raynaud's disease. The exit is usually caused by other complications. Perfect recovery may be obtained when gangrene has not already sacrificed any of the parts. Dr. George E. Brewer[4] has reported the case of a patient at the New York Hospital who developed symptoms of gangrene in both feet, which progressed so rapidly that both legs had to be amputated below the knees. The case was pronounced by Dr. Joseph Collins to be one of advanced Raynaud's disease. The

[1] Pester med.-chirurg. Presse, 1898.
[2] Clinical Journal, London, April 6, 1898.
[3] Pester med.-chirurg. Presse, 1898.
[4] Annals of Surgery, July, 1898.

patient's mental condition was one of partial dementia, and he was unable to give his name or age, and no history could be obtained other than that he had been out all night and frozen his hands and feet. It was supposed that the mental stupor was due to vaso-motor disturbance of the brain similar to that which in the lower extremities produced gangrene.

In this connection the case of Dr. C. E. Riggs is of interest,[1] for it is stated that Raynaud's disease represents a neurosis which is not perfectly clear, but has its foundation in the central nervous system. The patient was sixty-four years of age, one hundred and ninety-five pounds in weight, and for two years before the doctor saw her she had suffered from attacks of numbness of her fingers of half an hour's duration at a time. She was apparently well, when going up-stairs she was suddenly seized with numbness of the left hand and forearm, with loss of perception of heat and cold, and also complete paralysis of motion and sensation of the same. The hand was as white as marble. Next morning the finger-tips began to turn dark. Three days later she died. There were cardiac and renal complications. Unfortunately, the state of the pulse was not mentioned. The radial artery of the left arm was not atheromatous, and no thrombosis was discovered. Examination of the median nerve showed a simple parenchymatous degeneration; and of the spinal cord, from the fifth cervical to the sixth dorsal vertebræ, the posterior median columns showed degeneration and numerous capillary hemorrhages.

As to diagnosis, some authors regard Raynaud's disease in a wide sense, and consider as belonging thereto all affections whose essential symptom complex shows vaso-motor and trophic disturbance without regard to their origin; while others limit the picture of this affection much more sharply, and recognize the above-mentioned symptoms as Raynaud's disease only when they are without doubt caused by nervous disturbance and, in the absence of other causes, especially organic disease of the heart and blood-vessels.

Some writers suppose that erythromelalgia, or red neuralgia, is but the appearance of the second stage of Raynaud's disease

[1] Journal of the American Medical Association, December 10, 1898.

without the first. A little reflection might convince one that, if there be any relationship between them, they must be different members of the same family; for erythromelalgia usually occurs in women and does not show its peculiar change of color until the limb hangs down, when the arteries throb and the pain grows worse; it is aggravated by heat and eased by cold; it is asymmetrical, and never becomes gangrenous,—the opposite of what usually occurs in Raynaud's disease.

It would be quite as much to the purpose and equally wide of the mark to attempt to prove the identity of migraine and Raynaud's disease. Of two dozen articles which I have consulted on the latter, it seems to me somewhat remarkable that no one has pointed out the similarity between these two maladies, both of which present angio-neurotic phenomena much alike in the first two stages,—vaso-motor constriction followed by dilatation, and often affecting vision apparently in the same way. Rosenthal narrates the case of a hysterical girl in whom the migraine began with a sensation of cold in the fingers and toes of both sides. In a little while the face became pale. After the paroxysm the hands became warm and perspired freely, and the cheeks reddened.

Both Raynaud's disease and migraine are favorably influenced by massage.

Enough has been said about treatment in the preceding pages. It can be speedily summarized. When the local syncope and local asphyxia are slight and transient they require no treatment other than the avoidance of exposure to cold and excessive fatigue. Appropriate food and internal remedies, with plenty of fresh air for the anæmic and diabetic. Our very utmost endeavors should be used to improve the circulation, so that gangrene may not develop; and for this purpose Dr. Adolf Havas, in his article on this subject, already referred to, says that massage and remedial movements often act in a most excellent manner, preserving the patient from future danger. Such would certainly seem to have been the result in the first case reported in this article.

From the foregoing a few conclusions may be justifiable:

1. That when massage is of benefit in Raynaud's disease, it shows its effects very quickly.

2. These effects are improvement of the circulation, warmth, comfort, and suppleness.

3. The vitality of the tissues can not only be maintained and improved by means of massage, but even when destruction has begun it may be entirely recovered from.

4. As the beneficial effects of massage in Raynaud's disease are of a permanent character, it must, therefore, act not only upon the vaso-motor nerves of the affected parts, but also upon their central connections in the brain and spinal cord.

5. As the symptoms of Raynaud's disease would seem to be capable of affecting suddenly or gradually the vessels of almost every part of the human body, the most varied disturbances might thus find an explanation, whether they be sudden attacks of insanity, loss of consciousness, or asphyxia,—hemoglobinuria, colicky pains, or dead fingers, etc.

MOSQUITOES AND THE PROPHYLAXIS OF MALARIA.

ABSTRACTS FROM A LECTURE DELIVERED AT THE UNIVERSITY OF ROME.

BY PROFESSOR B. GRASSI,

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GENTLEMEN,—Until within the last year or two medical science has never been able to tell exactly how malarial infection originated. Now, after many futile labors, in the midst of which at times investigators despaired of ever being able ultimately to solve the mystery, we have arrived at a point where the solution of the problem seems at hand. Fortunately, this solution promises to be one that will enable us to eradicate malaria wherever it exists. The importance of this discovery cannot be overestimated. If throughout the length and breadth of the Italian peninsula, where nature smiles so maternally, this dread disease can be subdued, every acre of our prolific soil can be utilized, and a great future, far beyond even the most enthusiastic expectation, will open up for our country. Beyond this, the eradication of malaria will make the tropics habitable for all classes of men, and greatly favor the advance of civilization and culture throughout the earth.

Ten years ago a prominent member of the Italian parliament said, "If malaria comes from the soil, and this seems now to be indisputable, our struggle against malaria is vain. We are born in a country whose soil is by nature not a favorable one, and we cannot fight against fate." Only two years ago he repeated his former opinion even more forcibly. The problem of malaria he said remains scientifically unsolved. The utterance came at the very time when the observations were about to begin that have led to the solution of the malarial mystery.

The question, What are malarial fevers? can now be answered fully. They are nothing more than the expression of an infection produced by microbes. Malaria, like so many other diseases,—such as plague, typhoid fever, pneumonia, and so forth,—is microbic in origin. Under the name of malaria are comprehended three

varieties of fever, each produced by a special microbe. These three microbes are very similar one to another. They evidently belong to the same family, but they are at the same time very distinct from one another,—quite as distinct as are wheat and corn and rye.

One species of these three microbes produces tertian fever, a mild and benignant form of malaria; a second produces quartan fever; a third produces malignant tertian fever,—so-called pernicious malaria. The same person may fall a prey to all three of these microbes at the same time, and each will induce characteristic symptoms. As wheat produces wheat and corn yields corn, and never by any chance does one kind of grain produce the other, so the various malarial parasites reproduce their kind and each kind causes a special form of fever. One form of the malarial parasite can never be transformed into another.

While the malarial parasites are microbes in the sense that they are little living beings, they do not belong to the same family as the microbes which produce the other well-known contagious diseases. The plasmodium of Laveran has certain characters which make it clear that it belongs to the family of the protozoa,—*i.e.*, it is not a vegetable organism, as is the bacillus of tuberculosis or that of typhoid fever, but it is an animal parasite. If we examine one of these parasites shortly after its birth, we find that it resembles a little mass of colorless gelatinous substance. It penetrates into a red blood-corpuscle and proceeds to consume the material belonging to that cell. It remains without color except for certain blackish-brown granules that occur as the indigestible residue of the iron contents of the red globule. After it has attained a certain size, it divides into ten, twenty, or thirty little parasites, each of which in its turn invades a new red blood-corpuscle and proceeds to increase and multiply.

Certain recent discoveries go to show that the malarial parasites cannot continue their existence indefinitely in as simple a manner as this. After a certain time either a new vital impetus must be received, or else they cease to multiply, and grow old and die. This new impetus can come only from conjugation or fecundation. Until very recently we knew absolutely nothing of this phase of the reproduction of the malarial parasite. For a long time there had been noted, in the blood of human beings who had suffered from malaria for a long time, certain malarial parasites which, instead

of continuing the process of multiplication, became differentiated into elements usually of a semilunar form. These, so far as could be observed, always remained sterile in the body of man. Recent investigations point to the conclusion that these are the sexually mature organisms, which no longer propagate unless after conjugation. So long as they remain in the human system conjugation never takes place.

Before the marital union of the two sexes of the malarial organism can be celebrated they must wander far from their native place. Those who study biology find that nature uses marvellous means at times in order to secure the conjugation of the individuals of the same species. The astuteness she employs is often far beyond anything that the imagination of the most fervid romancer could ever construct. The case of the malarial parasites is certainly one of the most original among the means that are employed to bring together two beings that were intended for each other. The discovery of the road travelled by the malarial parasites on their wedding journey is one of the most important revelations made in recent years with regard to malaria.

The only place where the union of the male and female parasites of human malaria can take place is in the stomach of a peculiar variety of mosquito which bears the name anopheles. There are many insects which torment man by their stings. As is well known, they belong to very different families. Of mosquitoes alone there are more than twenty species known here in Italy. Only four of these, however, all belonging to the genus anopheles, present favorable conditions for the union of the malarial parasites. The other mosquitoes, notwithstanding their great similarity to anopheles, cannot serve this end. In the genus anopheles itself, only the females are capable of bringing the two sexes of the plasmodia together, since the males never sting and do not live by sucking blood.

When the female anopheles attacks a human being suffering from malaria, she sucks up together with his blood certain male and female elements of the malarial parasite which live in the blood. Once in the mosquito's stomach, the male rapidly moves towards the female and they unite. The new malarial parasite derived from this fusion of the two sexes is of an almost spherical figure. After a short time it becomes considerably longer than it

is broad,—acquires the form of a little worm. This organism moves freely in the stomach of anopheles, but after thirty or forty hours it proceeds to take up its quarters in the stomach wall. Here it remains for some time, becomes torpid, and after a while obeys in its turn the law "increase and multiply." It becomes large enough to be easily seen with an ordinary hand lens, and produces a number of germs which have the characteristic form of a little serpent. These descendants are very numerous: there may be ten thousand of them from a single parent. In order that they may remain within the body of the parent, this increases greatly in size. When the progeny has become mature, the capsule of the parent bursts, and the young malarial organisms of this sexual generation find themselves in the intestines of anopheles. They do not remain there long. If a specimen of anopheles be carefully examined, all of the new brood will be found gathered together in six little tubes which exist in the head of the mosquito. These tubes are the salivary glands of the insect. It is probable that these glands contain some substance which attracts the new malarial organism. In certain cases examination shows that the salivary glands of a single specimen of anopheles may contain millions of malarial parasites.

When anopheles stings, it injects into the puncture which it makes a portion of its saliva. If there should at the moment happen to be in its salivary glands any of the little thread-like malarial organisms just described, these will pass together with the saliva into the body of the human being stung. So it is that anopheles makes its inoculation of the malarial germ. These germs multiply within our blood, as we know, and, on the average, ten days after the inoculation there arises the train of symptoms that we designate malarial fever. A single sting of an anopheles mosquito whose salivary glands contain the malarial germ is amply sufficient to produce malarial fever in the human being.

And now the mystery of the goddess Fever, whom the Romans worshipped, is unveiled. Malaria does not exist; what exist are the parasites of malaria in man and in anopheles. Anopheles infects itself with malarial parasites from man and then conveys these parasites to other men. The various kinds of parasites of the different forms of human malaria develop indifferently in all the varieties of anopheles. If anopheles becomes infected with

the parasites of the benign tertian fever, the infection it conveys is that of benign tertian; if it is quartan originally, it remains quartan; and malignant tertian, in the same way, comes only from malignant tertian.

Nature cares nothing for the individual, but only for the race. In the human body the vast majority of malarial parasites die in the different stages of their development before they become sexually mature. Only those propagate sexually which by some chance are sucked up by anopheles; the others perish after a time. All the malarial parasites, male and female, that happen to be sucked up by other insects than anopheles pérish inevitably. Even in anopheles all the sexually immature individuals of the malarial parasite that happen to be sucked up likewise perish. Anopheles stings very freely most of the domestic animals and even occasionally birds. If in stinging it happens to inoculate malarial germs into these animals, the inoculation is without effect,—that is, the malarial germs die. It is true that certain other animals, especially birds, are subject to malaria. Avian malaria, however, and corresponding diseases in other animals are produced by parasites very different from those which cause malaria in man. They are not propagated by anopheles. Only man has the privilege of infecting anopheles. Only anopheles has the privilege of inoculating man. It is a sad privilege, but an incontestable one. Turn about is fair play, but may not be pleasant.

If you should conclude, from what I have said, that it suffices merely to bring the malarial human being and the anopheles mosquito together in order to propagate malaria, you would make a serious error. The malarial parasites develop within the body of anopheles only at a certain temperature. In man the malarial parasite finds a constantly favorable temperature, but the mosquito's temperature varies with that of the surrounding atmosphere. I have shown, by a series of experiments, that, when the temperature of the air is several degrees below 20° C. (about 68° F.), the malarial parasites, despite their sexual maturity, fail to unite in the stomach of anopheles. They die and are digested. It is clear, then, why, though in cold climates anopheles may be found and also human beings who have contracted malaria in warmer regions, malaria will not develop in the healthy people around them. Malaria flourishes especially in the warm seasons because

the high temperature favors the development of the malarial parasite in the mosquito and also makes this insect more numerous. Physicians have for many years recognized empirically the fact that the most suitable temperature for the development of malaria is about 20° C. (68° F.). Until recently we have not known the reason for this, nor why malaria exists with so much more virulence in tropical countries.

Before the malarial germs reach their new habitat in the salivary glands of anopheles the sting of the insect does not produce infection. Anopheles usually does not deposit in the puncture made by it all the malarial germs which it has at the moment in its salivary glands. To rid itself entirely of them it must sting a number of times, hence a single anopheles may infect several persons with malaria. In summer anopheles stings about once in every forty-eight hours. If it stings a number of malarial individuals in succession, it may infect itself with a number of malarial organisms. It may hence happen that the salivary glands of a single mosquito become filled, empty themselves, then are refilled, to be again emptied, and so on.

It is of fundamental interest to note that outside of the circuit of vital incidents of which we have spoken there is no possibility of continued life for the parasite of human malaria. It does not exist in any other surroundings than the body of man and the stomach and the salivary glands of the anopheles mosquito. Whenever it finds itself in any other medium, it perishes without fail. Other sucking insects, water, air, and the most diverse culture materials, all result inevitably in the death of the parasite. Malarially infected anopheles do not cause infection when eaten experimentally by man. The parasites in the body of anopheles, even the mature variety in the salivary glands, die and are digested in the gastro-intestinal tract. This shows beyond a doubt that malarial fevers are contracted not by way of the digestive tract, but only by subcutaneous inoculation.

It may be thought that there must exist some other media besides man and anopheles in which the malarial parasites can continue to live and multiply, but, notwithstanding a long series of experiments by myself and others undertaken for the purpose of elucidating this point, we have never succeeded in finding any trace of another medium. I cannot help believing that it is almost

absurd to expect to find another medium. The structure of the malarial parasite in its different stages of development is always extremely delicate. Its parasitic character—that is, its dependence on its host—is complete, and it is so absolutely without defence against unfavorable circumstances that it might almost be foretold without recourse to experiment that it could not live outside of its usual circuit. If the stomachs of mosquitoes very closely allied to anopheles are unsuitable for the development of the malarial parasite, as we now know, certainly any medium of a different kind will prove to be so.

Certain objections have been urged against the theory of the anopheles-borne nature of malaria. Some of these I shall take up and discuss. The dominant thought in the minds of many when they first hear of the mosquito theory takes the form of the question, "Is the new theory really founded on unexceptionable observations and proofs?" The word theory is no longer the proper one for this doctrine of the connection between malaria and the anopheles mosquito. The thesis has been proved and must be accepted as such. The preparations which show the life-history of the malarial parasite have been frequently and patiently examined by the greatest living specialists on the subject, who not only declare that the connection between the mosquito and malaria is absolutely proved, but also show the most perfect complaisance in accepting facts which at first seemed fantastic.

The experiments have been frequently repeated. The vital cycle, only hints of which were at first brought to light by the microscope, has been reconstructed point by point by experiments. Theory and zoological studies have been controlled by practical demonstrations. The healthy man has been seen to become ill of malaria in a non-malarial district as the result of being experimentally stung by anopheles. Man has been seen to contract malaria in a non-malarial season from the stings of anopheles mosquito which had been artificially infected and kept in a warm atmosphere. The healthy man has been seen to remain healthy after being repeatedly stung by anopheles which were known not to be infected. Anopheles has been seen to sting birds without infecting them. Other mosquitoes, it has been shown by a large number of observations and experiments, cannot infect man, and so on. In a word, the new doctrine is founded upon a granite base.

Assuming the truth of the mosquito theory of malaria, a second question that sometimes occurs is, "Did anopheles first obtain the malarial parasite from man, or was man first affected by anopheles?" A similar difficulty presents itself in many other biological questions,—*e.g.*, as to the priority of origin of the hen or the egg. This question might be asked in regard to all the parasites that exist in two hosts. For instance, did the tapeworms exist first in the ox or the hog, or in man? Did the echinococcus occur first in man and was the dog affected from him? It seems to me that there are a number of reasons for thinking that the malarial parasites first existed exclusively in the mosquitoes and only afterwards so developed as to be able to take up a new mode of life in man. There is a strong probability that the mosquito first infected man, but an absolute demonstration is impossible.

A third question that arises here is, "How can it be explained that healthy individuals have become infected with malaria in localities in which man has perhaps never before set foot?" We answer, first, that the fact of any such infection is not certain. Even if it were, however, it is a very simple matter to find a plausible explanation for the occurrence. Let us suppose an uninhabited locality where anopheles exists. A traveller who has never had malaria comes to the place with the usual retinue of guides and porters. There will almost certainly be among them some who, having had malaria previously, carry about with them the germs of the disease. The anopheles mosquitoes sting one or more of these individuals and become infected. A few days later they sting the traveller, and after the usual incubation period he falls sick. We may add that attacks of malaria in uninhabited places are really due not infrequently to relapses of the disease after a long interval and not to any reinfection. Besides, it has been noticed by travellers that malaria occurs, practically always, only in the neighborhood of inhabited places.

It would seem, for example, that malaria has been recently introduced into Caprera by visitors. Anopheles existed there before, but there were no malarial individuals to infect the insect. One sad day a garrison containing soldiers who had had malaria was stationed there. The anopheles mosquito became infected from these sufferers, and so this island also became malarious. Such facts are not easy to establish. So many possible factors must be

taken into account that the solution of the problem is very difficult. The mosquito theory has enabled us to explain the mystery better than any other.

A fourth question is, "How can the cases of malarial infection which occur in localities where there are no anopheles mosquitoes be explained?" Such cases if they really occur would be very difficult of explanation. In general, it is the custom to say that there are no mosquitoes in a place when but very few have been noticed. But these few, when a locality is really malarious, are practically all of the anopheles variety. They may be few in number yet easily accomplish a distribution of the malaria, for a single mosquito may infect several individuals. Up to the present time all the investigations of localities in which it was claimed that malaria occurred without anopheles in the vicinity to effect its distribution have proved the claim to be without foundation.

A fifth question is, "How can it be explained that in many localities where there is an abundance of mosquitoes no malaria is found?" This question is not difficult to answer. Where there are mosquitoes and no malaria, either there are no mosquitoes of the anopheles variety or the temperature is too low, or else there are no malarial human beings in the region to infect the anopheles.

A sixth question that has been asked is, "How explain the fact that malaria attacks most frequently those persons who expose themselves to the influence of water by taking cold baths and the like?" These conditions, it may be answered, somewhat favor the development of malaria by lowering the resistive vitality of the individual; ofttimes they expose him more surely to the sting of a mosquito; and, finally, such indiscretions frequently cause other fevers that are sometimes mistaken for malaria.

A seventh question is, "How is it that many localities become free from malaria, wholly or in part, by the introduction of good drinking-water?" It can only be said that the introduction of good drinking-water is usually accompanied by other changes in the sanitary surroundings of a place which make it more difficult for the anopheles mosquito to breed.

An eighth question is, "Is malaria propagated by anopheles alone? Is there no other method of infection?" Recent advances in biology have shown us that the sexual generation of the malarial organism takes place in anopheles. With this fact our knowledge

of malarial parasites becomes complete, at least in all essential particulars. It has been demonstrated that no other of the blood-sucking insects except anopheles furnishes a suitable place for this sexual generation. For the biologist, then, it becomes almost illogical to seek an explanation for the origin and propagation of malaria outside of man and the anopheles mosquito. Observations and experiments, as I have already said, exclude the possibility of water being the vehicle of malarial infection. Experiments have also shown that vegetables cannot be the medium of infection. Long experience has shown that the air itself is not infectious in a malarial locality. Men have been known to live in intensely malarial localities without taking malaria, provided they avoided the stings of anopheles. Besides, the anopheles theory of the origin of malaria explains practically all the peculiar phenomena of the disease which have hitherto been mysterious. For instance, it is now easy to understand why malaria does not occur when the temperature is below 18° to 20° C. (64.4° to 68° F.), why malaria is most easily acquired in swampy places, why the night air, especially just at nightfall, is most dangerous in malarial regions, and many other difficulties.

A ninth question that occurs is, "As it has been demonstrated that inoculating a healthy individual with a minimal quantity of blood taken from a patient suffering from malaria can directly convey the infection, it would seem that all the blood-sucking insects —fleas, bugs, mosquitoes, and even such animals as the leech—might easily convey malaria if they should happen to bite a healthy person while their sucking apparatus was covered with blood from the malarial patient; why, then, should we say that anopheles is the only agent in the transmission of malaria?" If the supposition were true, there would be many cases of malaria noted under circumstances that would at once determine the fact. We must remember, however, first, that these creatures do not permit their blood-sucking apparatus to remain moistened with blood for any length of time; second, they do not repeat their feeding without an interval of several hours, during which, it is well known, the malarial parasite will die if exposed to the air; finally, though these blood-suckers carry the infected blood containing the malarial parasites, they cannot inoculate it, because they lack the special organs necessary for this. It may be added that the minute quan-

tity of malarial blood shown to be capable of infecting a healthy man (it always amounts to a little more than one-tenth of a cubic centimetre) is yet an enormous amount as compared with that which would cling to the suctorial apparatus of a blood-sucking insect or even a leech.

Having discovered the cause of malaria, the next great question is that of prophylaxis. If anopheles mosquitoes are the propagators of malaria, how shall the human race defend itself against them? To answer this question properly, we must understand the life-history of anopheles. Anopheles deposits its eggs in water; from these eggs come the larvæ, which are transformed later into nymphæ and finally into adult insects. One or two days after it has acquired its wings the female mosquito begins its career as a blood-sucker; a little later it begins to deposit its eggs. The life-history of the mosquito occupies about fifty days. About thirty days after the eggs are laid the winged insect is developed, and twenty days subsequently the reproductive period sets in. Water is necessary for the development of anopheles, but not every kind of water will do. The insect propagates itself continuously only where there is water containing considerable organic material. Its favorite habitat, therefore, is a swamp overgrown with vegetation. The insect may exceptionally develop in small temporary pools of water destitute of vegetation, but after a generation or two under these conditions the race loses its vitality.

The first important safeguard, then, against the spread of malaria is the drainage of swamps and the prevention of the occurrence of pools of stagnant water. It is because these favor the reproduction and perpetuation of the anopheles mosquito that they have acquired the bad reputation which they justly bear in regard to malaria. Malaria does not come from the soil itself, and originates only indirectly from such stagnant water. Our ancestors were right when they attributed malaria to swamps, though they did not know the real connection between the swamps and the fever. They did not, however, sufficiently suspect the pernicious agency of small bodies of stagnant water, which may be quite as dangerous as swamps in the propagation of the disease.

One species of anopheles, the so-called anopheles claviger, prefers to take up its habitation in the vicinity of human beings. It passes the winter hidden in dark corners of our houses or in stables

or henneries. During other seasons, though it shuns the light less, these continue to be its favorite dwelling-places. It wanders away only when impelled by the reproductive instinct.

For prophylactic purposes it is important to determine the conditions which anopheles selects as the most favorable for its stinging expeditions. The moment of predilection is at twilight just before the obscurity becomes complete. The insects dislike wind, however, and so do not appear except on quiet evenings. Occasionally they sting also during the night if the weather is not too cold. Just at the break of day is another favorite time for obtaining their food. During the day—that is, whenever there is a bright light—they do not sting. Occasionally, however, in the rooms of a house where there is but little light during the day,—as, for example, where the shutters are thoroughly closed,—they may sting in the daytime. In a word, in a malarial region there is much more danger of catching the fever by night—and especially, as experience has shown, just at nightfall—than at any other time.

The most important part of the prophylaxis of malaria, however, consists in the care of human beings who are suffering from malaria. It is only from them that anopheles can obtain the parasites to distribute among the healthy. The sooner that human beings suffering from malaria can be cured the less is the danger of their serving as sources of infection for others. Quinine is, therefore, not only a specific curative measure for the individual suffering from malaria, but turns out to be also the most efficacious means for destroying the malarial germs, and hence preventing the propagation of the disease. It is important that patients suffering from malaria should be cured within the first five months of the year. During this time the mosquitoes, as a rule, do not become infected, owing to the low temperature of the air. A number of human beings, however, continue to suffer from malaria which they contracted in the preceding year, and when the weather becomes warm they will infect the mosquitoes.

Another important prophylactic measure is to keep anopheles from feeding on malarial patients. If mosquito-netting be placed over the windows and doors, and even here and there in the corridors of houses in which malarial patients live, we may be able to prevent the mosquitoes of a neighborhood from becoming infected.

Whenever a healthy person is obliged to sleep unprotected by mosquito-netting in a malarial neighborhood, he should burn in his bedroom some of the many powders that have the reputation of driving away the insects. Certain of these, as pyrethrum, are known to be effective. Whenever any one is obliged to sleep in the open air, he should protect himself with pieces of mosquito-netting attached to trees, or to poles after the manner of a tent.

There is every reason to believe that our present knowledge of the manner in which malaria is distributed will eventually enable us greatly to limit the spread of the disease. It is not probable that we shall ever be able entirely to destroy the anopheles mosquitoes, but we may diminish their number very much and so lessen the amount of harm that they can do. Malaria will in the near future become as rare as have many of the infectious diseases which were so justly dreaded, and often caused such wide-spread mortality, before the discoveries of modern medicine robbed them of their terrors.

Symposium on Genito-Urinary Diseases

PART II.

TREATMENT OF CHRONIC GONORRHŒA, OR GLEET.

CLINICAL LECTURE DELIVERED AT THE BROCA HOSPITAL.

BY ALEXANDRE RENAULT, M.D.,
Physician to the Paris Hospitals.

GENTLEMEN,—In view of the many cases of gleet that you are constantly seeing treated among the out-patients of our wards, it seems to me that I shall be rendering you a service by giving as succinctly as possible a general outline of the methods of treating this very common and very troublesome disorder.

But, first, what are we to understand by chronic gonorrhœa? At the end of what length of time does an acute case become chronic? The answer is, in about three months. An acute case that we have not been able to abort—and such is the rule—ought to be cured in six weeks. To this should be added two weeks more of care, in order to be sure of the cure. Then if we again add another month for the many patients who either neglect to follow in detail the treatment prescribed or lack the will-power to adhere to the regimen that the disorder requires, we get a total of three months, after which period the disorder becomes chronic.

The principal symptom of this condition is usually indicated by the patients themselves, whose first remark is that every morning on awakening they see a drop at the end of the penis. At the least excess this quantity increases. During the daytime there is also a slight secretion, but unaccompanied by pain. You must then

complete the diagnosis by looking for two other symptoms, fully as important as the first one.

First see how the patient urinates, to ascertain whether there is any probability of the presence of stricture. Ask him if he urinates as easily as formerly, whether the stream is as full as ever and is projected as far, and whether the last few drops are not apt to fall at his feet. When possible try and get the patient to urinate in your presence, though, as you are aware, many nervous men cannot possibly accomplish this. In the latter case you must explore the canal with instruments, as no treatment will cure the gleet so long as the calibre of the urethra is not normal.

The third sign will be found in the urine itself. There is little use looking for the drop of which the patients complain; as a general thing, no amount of pressing on the canal will produce it; in fact, the patients themselves say that it is there only on awakening in the morning. The most that you can find in some cases are one or two almost imperceptible filaments extending from one side of the meatus to the other, indicating deep and chronic inflammation with viscous secretion gluing the two sides together. The mucous membrane will often be found to be dark red or purplish, instead of the normal pale pink or even pallid color. Should you wish to see the drop, you must arrange to visit the patient early in the morning before he passes water.

But as regards the third symptom. Get your patient to urinate the first half of his urine into one vessel, and the latter part into another, telling him to be sure to completely empty his bladder and canal. This is important; the last contractions should be vigorous in order to entirely empty the contents of the glands of the posterior urethra.

In the first vessel you will see filaments and flakes floating in the liquid. These are occasionally found in the urine of healthy persons, but in them such a condition is very unusual, and when it exists the difference in appearance is striking. In the normal state the filaments are few in number, visible only by transparence, and thin; in gleet they are long, white, and opaque, and many of them fall to the bottom of the vessel. In the second glass you may also find filaments and flakes, but as a rule they are not present.

When there are abnormal elements in the second glass the diagnosis of the seat of the gleet can be at once made, and you may

be positive that the disorder has spread to the membranous and prostatic portions of the urethra, the filaments in the last few drops of urine having been squeezed out of the glands of the deeper urethra by the contractions of the muscular tissues surrounding the membrano-prostatic zone.

Even should the second half of the urine be clear, we are, unfortunately, not warranted in assuming that the disorder is confined to the anterior urethra alone. In most cases the first part of the urine carries away all the abnormal elements in the canal from the neck of the bladder to the meatus. If, during the acute phase of the disorder, the patient suffers from a complication such as cystitis or epididymitis, you may, even should the latter half of the urine contain no filaments, still be sure that the posterior urethra is affected; otherwise, the only factor to rely on is the duration of the case. The rapidity with which the gonococcus invades the canal is not yet determined and appears to vary; but from what we already know it is safe to say that, with very few exceptions, any case of gonorrhœa that has lasted two months has extended to the entire urethra.

There is a practical method of ascertaining positively whether or not the deep portion of the urethra is affected. After taking the usual antiseptic precautions, introduce a soft red catheter (No. 10, French) to the end of the anterior urethra, where it will be stopped by the contraction of the membranous sphincter. Then, with a sterilized syringe and boric solution, freely irrigate the anterior urethra. This irrigation will carry away all the filaments of the anterior portion, and should be continued until the liquid comes out absolutely clear. Then ask your patient to urinate in a glass; if the urine contains filaments and flakes, it is certain that they come from the posterior urethra.

The functional disturbances that accompany the above-mentioned objective symptoms are of secondary importance only. They corroborate the physical signs, but in themselves are useful only as an indication of deep urethritis. This is also practically true of bacteriological exploration of the secretions of the canal.

The diagnosis once made, you should proceed as follows. If there is no stricture, begin the treatment that will be given further on. If there is a stricture, you must first dilate it, since, as I have already observed, no treatment will succeed until this obstacle is

removed. Furthermore, the mere dilatation of the stricture may suffice to cure the gleet. This result is not easy of explanation; but you should know that the dilating catheters not only permit drainage and restore the canal to its normal calibre, but have an effect on the mucous membrane not unlike massage, freeing the glands from their morbid contents, modifying this membrane little by little, and increasing its vitality.

Before undertaking to dilate a stricture, you must have the required outfit. This consists of at least two exploring catheters (*bougies à boule*), Nos. 16 and 18, and an entire set of flexible conical bougies, from No. 4 to No. 24. Those below No. 4 cannot be passed through a tight stricture, which consequently must be treated surgically.

All these instruments are to be kept perfectly aseptic, and for this purpose tri-oxymethylene, a powder whose chemical composition is very similar to that of formol (which cannot be used owing to its being a liquid), is the best agent. The most practical method of using this powder is to keep all urethral instruments in a long glass tube, at the bottom of which is placed the powder in a sachet, while the top is fitted with a plate pierced with a suitable number of holes of proper dimensions through which the catheters are passed and from which they hang, this plate in turn being covered by a closely fitting cap that hermetically seals the tube. Such tubes cost but little. The sachet of powder should be renewed at least every two months.

You must begin by cleaning your hands and the patient's penis just as you would for a cutting operation, irrigating the canal with a warm boric solution by means of a glass syringe, which can be kept perfectly aseptic by lying constantly half filled with a 1 to 1000 bichloride solution.

Withdraw from your tube a bulb-explorer No. 18, which is practically the size corresponding to a normal urethra, and see whether or not there is a stricture present. You must first wipe off the catheter with warm boric water to free it from tri-oxymethylene vapors, which are irritating, and then dip it in carbolic oil 1 to 20. Pass the instrument gently into the urethra, and, if the canal is normal, it ought to reach the bladder without hindrance. It may, however, be stopped short at the membranous sphincter; in this case wait a few moments, drawing the explorer back a little.

Distract the patient's attention by some question, or get him to breathe deeply and relax his muscles. As soon as the spasmodic contraction of the sphincter ceases the catheter will pass through without the slightest trouble. Another way of overcoming this difficulty is to apply cocaine and vaseline (1 to 20) to the end of the instrument, and bring it into contact with the resisting sphincter for a moment or two. If, however, the obstacle proves to be a permanent one, you have to do with an organic stricture.

This is the way the case presents itself when the stricture has occurred at the point of union of the two portions of the urethra. But in practice it is usual to find, before your instruments reach that depth, points of coarctation much nearer the meatus, which at once give you an exact diagnosis.

Knowing that you have a stricture to dilate, try bulb-explorers of decreasing numbers until you find one that will pass through it. Replace this instrument by a conical bougie of the same number, leave it in place for from two to three minutes, then withdraw it gently, and your first day's treatment is over.

After that you should see your patient every two days, unless the treatment is followed by pain while urinating. In such a case you had better wait until all pain has disappeared before continuing the dilatation. At each seance you should insert two bougies,—the one you used last at the preceding visit and that next in number. Thus, supposing you begin with No. 8, at the next visit you should use Nos. 8 and 9, at the next Nos. 9 and 10, and so on, each time with the same antiseptic precautions as mentioned above.

The urethra should be dilated in this way as far as No. 22; it will then have a diameter of at least seven millimetres, the normal being about six, and this will be quite sufficient.

The dilatation can also be done with the metallic bougies called Béniqués, each number of which is one-sixth of a millimetre larger in calibre than the preceding one. Of these you can use three at each seance, instead of two of the flexible kind, but they are not adapted for small strictures. It is well not to use them below No. 32, which corresponds to No. 16 of the flexible bougie, or ordinary French scale.

Having dilated the stricture, you must keep the ground you have won. Pass the highest number of bougie used every month for, say, six months, and after that once or twice a year.

It is possible that when the dilatation is finished you will find that the gleet is cured. The "morning drop" disappears, and only a few filaments will be left in the urine. Such, however, is, unfortunately, not the rule. The dilatation visibly improves the situation, but the canal does not dry up definitely. You must then employ either the internal or the external means at your disposal to cure the catarrh of this mucous membrane.

You had far better commence with the internal treatment, which is easy both for you and the patient, and can do no harm.

The mixture of copaiba and cubebs in definite proportions is unquestionably the best preparation, but if you desire to succeed with this method you must give your patient minute directions about it. Physicians frequently prescribe this remedy in a paste, telling the patients to make so many pills of it and take them at certain intervals. This should not be left to the patient, but to the druggist. Prescribe as follows:

℞ Copaiba balsam,
Powdered cubebs, āā ℥i;
Calcined magnesia, q.s. to give proper consistency.
Mix and divide into sixty capsules.

Each capsule contains one-half gramme of both of the active substances. The patient should at first take six a day; in forty-eight hours increase to twelve a day. Then, if the discharge is not modified in a week, increase to eighteen. The remedy should be taken in three doses, immediately before meals, as at that time the stomach will better tolerate the treatment. If diarrhœa occurs, the druggist can add to each capsule from 0.10 to 0.15 gramme of bismuth subnitrate. Diarrhœa indicates that the medicine is eliminated by the intestine instead of by the urinary tract, which is unfortunate.

The quantity of this mixture that a patient can tolerate varies with the individual. The majority of patients stand from six to nine grammes of each very well; some can even take twelve grammes without discomfort, but this requires a digestive tube in remarkably good condition. These large doses render the administration of this preparation in capsules somewhat difficult, as each capsule contains but a small quantity and consequently the patient has to consume a number of them. Instead of the mixture I recom-

mend, you can give the copaiba pure, but in that case you are obliged to administer it in capsules on account of its very disagreeable taste.

Copaiba alone may stop the discharge, but I prefer the mixture given above, which does not upset the digestion any more than the balsam alone, and which unquestionably is more efficacious. As for the cubebs given by themselves, they are even less active than the balsam alone, and therefore I do not advise them.

There, are, however, patients who can take neither the mixture nor the balsam alone. To these you may give the oil of santal in capsules of 0.25 each (gr. iv). Begin with six a day, and increase gradually up to sixteen, which equals four grammes of santal oil in twenty-four hours. Should the patient object to these capsules, you can give an equal amount of the drug in a syrup.

In whatever form the remedy is taken, it is very important that the dose should be given just before a meal. When mixed with the food it is much better tolerated by the stomach and can more readily be digested. Still, when the dose is raised to sixteen capsules per diem, the last four can be taken at bedtime with a little milk, allowing two hours to elapse after dinner.

You doubtless know that with some patients the passage of santal oil through the kidneys gives rise to violent pains in the small of the back. This accident, which, happily, is very uncommon, will necessitate your stopping the medicine for a while, to take it up later in smaller doses.

Should the santal oil fail, you can try essence of turpentine in capsules or syrup in similar doses. But this remedy is less active than the one before mentioned, and I do not advise you to expect good results from it in cases in which the foregoing preparations have failed.

Another thing that you must impress upon your patients is not to stop the preparation as soon as they find that the discharge has ceased. This may be said to be the case when, on awakening in the morning, after having been at least seven hours without urinating, the patient finds that the sides of the meatus are not stuck together, and that no drop appears at the opening of the canal when the foreskin is brought entirely back and then forward again, with pressure on the urethra. There must be neither partial nor full erection when this test is made, otherwise the results may de-

ceive you; you may find the meatus stuck together by the mucus secreted as a consequence of the erection; and, in addition, on separating the two sides of the meatus you may also find this mucus in the canal, and resembling in its opaline tint that of morbid origin.

Your test should be closed by getting the patient to urinate in a glass. I have just explained to you what the characters of normal filaments are; if in this urine the filaments are opaque and drop to the bottom, be on your guard. There is still some inflammation left which a stimulant of any sort might very easily bring to an acute phase once more. In such a case you should tell your patient to continue the remedy for ten days after you have decided that the disorder is cured, lessening the dose; and this he should do whether or not the urine contains filaments, for the purpose of consolidating the cure.

Fully as necessary as the internal treatment is the diet that the patient must be made to follow. This is particularly true of chronic gonorrhœa. I never allow any drink except milk, milk and water, or water, and I advise him to drink only when absolutely necessary, so as not to increase the flow of urine. Vinegar, mustard, spices, and asparagus should be forbidden, as well as all violent forms of exercise (long walks, bicycle and horseback riding, fencing, etc.), on account of the danger of producing epididymitis.

This diet, which should be begun at the same time that you prescribe medicinal treatment, must be kept up fully ten or fifteen days after all remedies have been discontinued, and your patient should resume the ordinary occupations of a healthy man by degrees only. Beer, champagne, and sexual intercourse should not be indulged in for at least a month after all discharge has ceased. Attempted sooner than this, sexual intercourse may have no bad result, but I consider the experiment hazardous.

THE TREATMENT OF URETHRITIS IN THE MALE.

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Broad general principles, not the routine application of any one method, should govern us in the treatment of urethritis. That there cannot be one fixed method is fundamental and elementary. This should become axiomatic when we consider the etiology and comprehend the pathology based upon it and upon the anatomy and physiology of the genito-urinary apparatus.

Cases of urethritis differ according to the nature and virulence of the infection, the region and structures invaded by and involved in the inflammatory process, the stage of the inflammatory process at the time when the patient presents himself, and, finally, the personality of the patient himself,—his power of resistance and his ability to carry out instructions faithfully and intelligently. The personal equation of the patient enters into this disease as into any other. What we actually treat is a patient with urethritis, not a "case of urethritis" complicated by a patient.

By all these factors, therefore, is the treatment of urethritis influenced and determined: the etiology, the virulence of the infection, the pathological condition, the stage of the inflammatory process, and the patient.

Consequently, the treatment must vary. Thus, in acute urethritis we may or may not put the patient to bed in addition to regulating his regimen; we may or may not prescribe a hand injection of protargol solution at once, and if we do, it may be a one-half instead of a normal strength (one per cent.) solution; we may or may not, in addition, irrigate the penile urethra; we may or may not practise irrigations alone.

The only routine that may be said to exist in the treatment is in the method of approaching a case and in the general directions that should be given every patient having this disease. The patient's general condition must be taken into account. We note whether he is anæmic, whether disposed to oxaluria or the uric acid diathesis, and we prescribe accordingly. We see to it that his diet is so regulated as to avoid highly spiced and stimulating articles. We must get him to reduce his alcohol and tobacco, especially the former, eventually stopping it altogether, and we must limit his coffee-drinking to one cup a day, preferably taken at breakfast. He must be directed to drink freely of either plain water or an alkaline water between meals. Any tendency to constipation must be corrected. He must wear a suspensory bandage and be taught to dress the penis properly, carefully avoiding anything that will interfere with the free outflowing of the urethral discharge. He must be cautioned as to the danger of infecting his eyes. All sexual relations, passive as well as active, must be forbidden.

ACUTE ANTERIOR URETHRITIS.

In this form of the disease we have to deal with either a simple catarrhal or a purulent inflammation of microbic origin, in the vast majority of cases, affecting the mucous membrane of the urethra and productive of more or less damage to one or more of the layers and structures composing it. Disregarding the former,—the relatively rarer cases of simple catarrhal inflammation of the urethra due to traumatic or chemic irritation,—and confining ourselves to the purulent form, we summarize the stages and the indications to be met as follows:

(1) *The acute stage. The eradication of etiologic micro-organisms.*

(2) *The subacute stage. The termination of the catarrhal discharge.*

(3) *The chronic stage. The repair of damaged mucous membrane.*

(1) *The Acute Stage.—The Eradication of Etiologic Micro-Organisms.*—In all varieties of acute infections of the urethra, whether purely gonorrhœal, or due to a "mixed infection" by the gonococci and one or more of the other pathogenic micro-organisms, or to these last alone,—in all, a hand injection of a one-half to one

per cent. solution of protargol is thus far the most efficacious means at our disposal for this indication. Its use should be begun as soon as the disease has manifested itself: in the prodromal stage if possible, provided the microbic character can be demonstrated in the given case. While efficacious in all these varieties of acute anterior urethritis, it is in the gonorrhœal form that its promptly germicidal properties are most marked. It is almost a specific in these cases. Under the use of such a solution, at once placed in the hands of the patient upon the first appearance of a discharge containing the gonococci, we have seen the gonococci disappear from the pus in two days. During that period the quantity of discharge was increased somewhat, when it began to subside, and by the sixth day only the "morning drop" remained. In a case in which the patient presented himself nine days after the discharge had begun, the gonococci disappeared and the free discharge almost ceased in four days. Similarly in a third case, seen fourteen hours after the discharge began, gonococci were not found after the fourth day, and the discharge had diminished to a milky "morning drop" before the eighth day. Such cases might be multiplied, but the above will suffice to illustrate the point.

As a matter of course this comfortable and desirable condition is not permanent at this period. The cessation of an urethral discharge under these conditions never means that the patient is *cured* of the disease. Sooner or later a relapse will occur if, under that erroneous assumption, we now discontinue the protargol solution. The gonococci that penetrated to the deeper layers of the mucous membrane during the period of incubation and there multiplied, have not been destroyed; they have been inhibited only. Left to themselves they again become virulent, and the reappearance of a discharge laden with gonococci is the result. It is, therefore, necessary to continue the protargol hand injection from three to four days after the cessation of the discharge, and then gradually to diminish the frequency of its use, at the same time being watchful for the reappearance of the gonococci. Experience soon enables us to judge when the protargol solution may be discontinued. Nevertheless, the catarrhal discharge usually present by this time should be examined every few days for gonococci. Their reappearance is the signal for instructing the patient to resume the injection.

The syringe recommended for hand injections is the ordinary,

cheap, quarter-ounce syringe with glass barrel and soft-rubber tip, or the blunt-pointed quarter-ounce syringe of hard rubber. The patient should be instructed how to use it, and impressed with the importance of scrubbing it thoroughly each time after having had it in contact with the meatus. The injected solution of protargol should be retained in the urethra five minutes.

When the inflammation excited by the infection is of high grade, as evidenced by the red and œdematous condition of the meatus, antiphlogistic measures are indicated in addition; much more so should there be general turgescence of the penis. In such cases it may be wise, if not imperative, to put the patient in bed also. A warm, moist compress loosely wrapped about the penis and surrounded by a layer of rubber tissue, hot sitz-baths or merely soaking the penis in hot water several times daily, fulfil the indications as regards the external surface; hot solutions for copious irrigation from the meatus fulfil them as regards the urethra, if the patient can bear irrigations. For the purpose a normal saline solution, a saturated solution of *chemically pure* boracic acid, a one-half-strength Thiersch solution, a 1 to 5000 solution of potassium permanganate, or one of mercuric bichloride 1 to 30,000, may be employed. The temperature should be as high as the patient can comfortably bear. The pressure of the fluid should be low, in order to confine the irrigation to the penile urethra, and to avoid undue pain. Preferably the irrigation should be given from a Janet or other similar syringe, so that the pressure shall be under control of the surgeon's sense of touch. If an irrigator be used, the reservoir should hang at a previously determined, moderate height above the patient's hips, and the fluid should be delivered from a blunt glass nozzle gently applied to the meatus.

The value of irrigation does not lie in the medicament alone, but in the moist heat brought to bear upon the engorged mucous membrane. Through the reduction of the inflammation thus effected (which means a reduction of the abnormally increased blood-supply in the mucous membrane) they rob the etiologic micrococci of the conditions which favor their growth, thereby, as it were, starving them to death. A germicidal agent, such as protargol, by its prompt destruction of the gonococci, cuts short the inflammatory process excited by them and moderates the course of the disease; on the other hand, irrigation destroys the gonococci secondarily by first

reducing the grade of the inflammatory process, thus indirectly and therefore less promptly attaining the desired end.

In this way is explained the value of irrigations, whether used alone, as they sometimes are in certain cases of streptococcus infection in which protargol seems not to effect the process, or as an adjunct to the latter in cases of pure or mixed infection. Relied upon alone in the last-named varieties of urethritis, irrigations do not yield as satisfactory results as do the hand injections of protargol when used according to directions.

In most cases the patient can carry out the "irrigations" sufficiently well himself if he be provided with tablets of mercuric bichloride, one of which dissolved in a fixed quantity of hot water will yield a 1 to 30,000 solution. Of this he is to inject twenty or thirty syringefuls one after the other two or three times daily, using his quarter-ounce syringe for the purpose. When necessary these "irrigations" may be supplemented by the surgeon.

(2) *The Subacute Stage.—The Termination of the Catarrhal Discharge.*—The micrococci having disappeared, the inflammatory signs having in large measure subsided, and the urethral discharge having become muco-purulent, perhaps mucoid, and possibly limited to the "morning drop," the treatment as outlined above is continued four or five days longer and then gradually and tentatively withdrawn while a hand injection of a mildly astringent solution is being substituted. From twice a day the protargol solution is first reduced to once a day and then discontinued altogether after another interval of from four to ten days, depending upon the case; while the astringent solution is simultaneously begun at the rate of once a day and so continued until increased to twice a day when the other treatment is discontinued. The best astringent injection at this stage is the following:

℞ Zinci iodidi,
Zinci chloridi, aa gr. i;
Aquæ destillatæ, q. s. ad f℥iv.
M. Sig.—Use warm, as directed.

It should be warmed before use by immersing a syringeful in hot water. It should be retained in the urethra one minute. As already noted, the mucoid discharge present will bear microscopic examination every four or five days. If cocci reappear recourse should be

had to the original treatment, or the protargol solution resumed in connection with the astringent.

(3) *The Chronic Stage.—The Repair of the Damaged Mucous Membrane.*—Here silver nitrate in solution is by far the best agent because safest and most efficient. It is brought into contact with the mucous membrane by means of either the Ultzmann syringe or the Bangs syringe-sound, or by cotton swabs through the endoscope. 1 to 1000 (one-half grain to the fluidounce) is a commencing strength for instillation with the syringe-sound; this is gradually increased to two or three grains to the fluidounce. A solution of five grains to the fluidounce is the first for application through the endoscope. Stronger than ten grains to the fluidounce is not advised. Generally these instillations and applications are begun as soon as the discharge has become mucoid; it is not necessary to wait for the total disappearance of the "morning drop." Hence their employment dovetails with the astringent hand injections as the latter did with the protargol, *excepting as to frequency.* The instrumentation necessary to their use, together with the stimulating effect of the silver nitrate solution, excites a reaction in the mucous membrane. There will be more or less increase in the discharge and possibly some smarting on urination for from twelve to twenty-four hours afterwards. This reaction must be allowed to subside and the tonic effect of the treatment must be obtained before the mucous membrane should be subjected again to this unavoidable excitation. It is important to avoid over-stimulation of the urethra; therefore no form of local treatment by instrumentation should be repeated oftener than once in five days,—in some cases only once in seven days. (To this rule there is an exception, which will be mentioned later.) As a matter of course, all instrumentation of the urethra should be practised with the utmost gentleness.

The Bangs syringe-sound has a great advantage over the Ultzmann syringe in that it gently opens out the folds in the urethral mucous membrane for the reception of the twenty or thirty minims of silver nitrate solution that are instilled into the urethra from the syringe when the point of the sound has reached the objective spot. By means of the endoscope damaged points, *e.g.,* granular patches, zones or areas of chronic congestion, and superficial erosions, are sought out and applications made directly to them.

We have now to determine when the astringent hand injection

may be discontinued. The aim is to dispense with it as soon as possible. We test the question by reducing the frequency of its use; finally stopping it altogether and noting the result. If the catarrhal discharge increases to more than the "morning drop" and

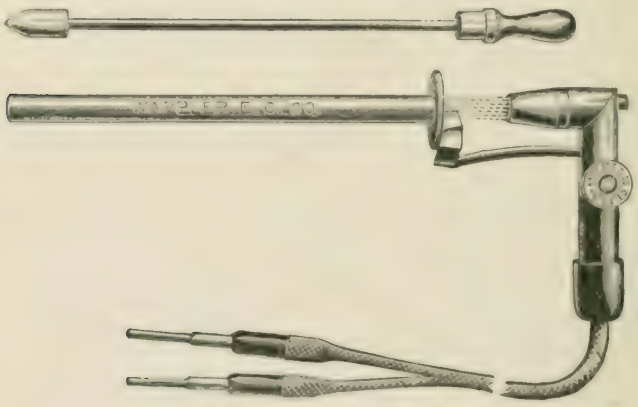
FIG. 1.

The Bangs syringe-sound consists of a *syringe*, the distal end of which fits into the proximal end of the *sound*. Traversing the length of the sound and opening at its beak is a tube of small calibre, through which the solution in the barrel of the syringe is discharged when the piston is pushed home. The sounds are graduated in size and marked according to the French scale.

persists thus for three days, we have the patient resume the astringent. After an interval of a few days we again apply the test.

In much the same way we begin now to taper off the instillations and applications that have been continued steadily meanwhile. When the "morning drop" has entirely disappeared and

FIG. 2.



The improved Otis endoscope.

the urine shows only a transparent mucous thread suspended near the surface or, at most, one or two light shreds or flakes slowly settling towards the bottom, all treatment should be discontinued and the patient told to report in ten days for inspection. When finally dismissed he should be cautioned that he is liable to a return of the catarrhal discharge at any time within six weeks if he dis-

obeys any of the general directions given him at the outset. The duration—*i.e.*, the time under treatment—of an initial, uncomplicated, acute anterior urethritis, seen early in its development, is exceptionally three weeks. Usually it is from four to six weeks, depending upon the several factors previously enumerated. To quote Neisser: "It is not the rapidity but the *certainty* of the cure that is sought."

As to the complications of acute anterior urethritis, the three most likely to protract its course are peri-urethral infiltration, folliculitis (possibly resulting in a peri-urethral abscess), and cowperitis.

Chordee is a symptom of the acute stage that marks a cellular infiltration of the submucous tissue. It calls for antiphlogistic means locally, especially hot irrigations, and for such drugs as the bromides and antipyrin internally. The diet must be of the blandest. The patient should sleep lightly covered. In the chronic stage irrigations, gradual dilatation, and topical applications with the silver nitrate solutions are indicated.

Mild folliculitis usually escapes detection during the acute stage. There are several methods of treating it in the chronic stage, all having for their object the mechanical expression of the morbid contents of the follicles and the introduction into them of a medicament. Thus, irrigations with a 1 to 30,000 mercuric bichloride solution under some degree of pressure; distention of the penile urethra with a 1 to 1000 solution of silver nitrate, a one per cent. protargol solution, or a five per cent. solution of iodoform in sterile sweet almond oil, together with massage of the urethra from the meatus backward by means of gentle pressure with the fingers; the passage of a moderate-sized sound lubricated with Finger's ointment and massage of the urethra against the sound. The formula for Finger's ointment is,—

℞ Iodini, gr. v;
Ol. olivæ, ʒss;
Potassii iodidi, gr. xxx;
Lanolini, q.s. ad ʒi.
M. Sig.—Finger's ointment.

Peri-urethral abscess and cowperitis call for antiphlogistic measures, applied externally, and incision as soon as fluctuation is present.

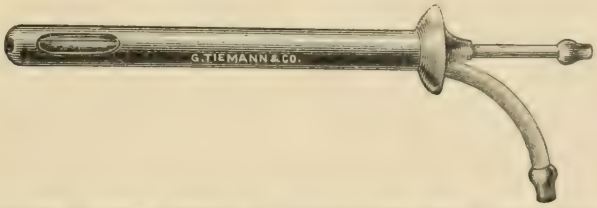
ACUTE POSTERIOR URETHRITIS.

As soon as the posterior urethra has become invaded it is well to put the patient to bed, if possible. Fortunately, most of these cases are marked by only a day or two of very slight frequency and possibly urgency of micturition, which do not make this degree of rest imperative, and which do not call for any special treatment beyond the general measures already enumerated. Again, there is a second class of cases characterized by symptoms of sufficient severity to require relief. This may be obtained by rest and by irrigation from the meatus gently and yet with sufficient pressure to carry the fluid into the bladder. This manœuvre can be facilitated by having the patient's thighs slightly flexed. The irrigating fluid may be any one of those given under acute anterior urethritis. Between two and three ounces should be injected in this way into the bladder, and the patient then allowed to stand up and void it. Finally, there is a third class of cases,—namely, those in which the symptoms are a marked frequency and urgency of urination with tenesmus and blood at the end of the act. These patients voluntarily go to bed. For local treatment we at once instil from fifteen to twenty minims of a 1 to 1000 solution of silver nitrate into the deep urethra, disregarding all other conditions. If the patient can be kept in bed under control, stronger solutions, up to five grains to the fluidounce, may be given. The relief that after a few hours follows the temporary aggravation of the symptoms is surprising. For this instillation we may use a soft-rubber catheter, the Ultzmann syringe, or a Bangs syringe-sound of 20 French. Unless the surgeon is skilled in the passing of urethral instruments, and unless he is prepared to use the utmost gentleness and caution, a smooth, blunt-pointed, sterile, soft-rubber catheter should be the instrument chosen. Naturally, the penile urethra is exquisitely sensitive, being itself acutely inflamed; it may have to be anæsthetized with a two per cent. solution of eucaine before the introduction of any instrument is possible. If the first instillation proves successful, it may be repeated every second or third day should the indications return, this being the one exception to the rule regarding the frequency of instrumentation during the treatment of urethritis.

As to the complications of acute posterior urethritis, urethrocystitis, epididymitis, prostatitis, and seminal vesiculitis are the immediate ones. To go into a detailed description of the treat-

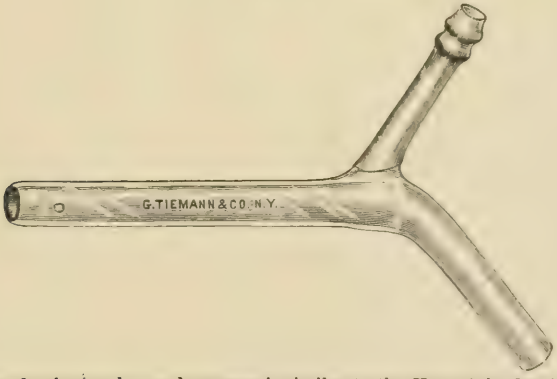
ment of each of these would carry this paper beyond the prescribed limits. Briefly, they are to be treated according to well-known general principles,—rest in bed, diet, and regulation of the bowels. Urethro-cystitis is managed as is acute posterior urethritis of the first and second grades. For epididymitis, perfect support of the

FIG. 3.



The Kemp tube for rectal irrigation is a double-current tube of hard rubber or of metal. The inflowing current is delivered through the small opening at the end of the tube; the outflow is through the two large "eyes" near the end of the tube and thence by way of the outlet tip, shown as curving downward in the cut.

FIG. 4.



The Chetwood tube in plan and purpose is similar to the Kemp tube, but is made of glass, and the inflowing current is delivered through small openings on the side of the tube, while the outflow is by way of the large opening at the end.

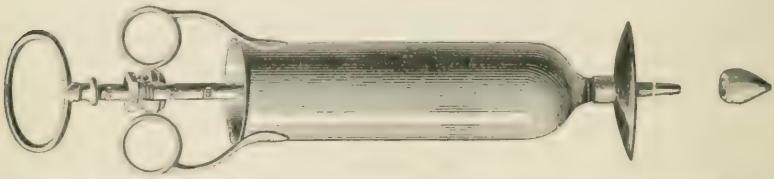
testicle, counter-irritation, and the ice-bag or poultices are indicated. Acute prostatitis and seminal vesiculitis should be carefully watched for signs of suppuration. To avert it we may apply cold, preferably by means of a rectal psychrophore, or heat by means of hot rectal irrigations with a normal salt solution heated to from 100° to 120° F., administered through the Kemp or Chetwood tube. If the introduction of the instrument cause excessive pain, this treatment should be discontinued and cold or heat applied externally. The vast majority of these cases go on to resolution.

If, however, an abscess forms, it should be incised perineally to avoid a possible rupture into the bladder or rectum, or both.

CHRONIC URETHRITIS (ANTERIOR AND POSTERIOR).

The management of a patient with this form of urethritis and the treatment of the local conditions are practically the same as in the third, the chronic stage of anterior urethritis, preceded by a critical examination of the urethra and its accessory parts for contributing causes and complications, such as redundant prepuce, phimosis, infected preputial follicles, narrow meatus, stricture, chronically infected urethral follicles, chronic cowperitis, prostatitis, and seminal vesiculitis.

FIG. 5.



The 160 c.c. Janet syringe, with detachable shield and blunt tip of hard rubber.

An infected prepuce had best be removed by circumcision. A narrow meatus may require meatotomy. The treatment of stricture, whether organic or spasmodic, is too broad a subject to admit of discussion here. Briefly, organic strictures should be dilated whenever possible and, at the same time, the surrounding congested, perhaps granular, mucous membrane should be treated by topical applications of silver nitrate solutions. Should dilatation fail, urethrotomy may be performed. Spasmodic stricture calls for restoration of the urethra in general to as near the normal state as possible and careful attention to the quality of the urine. Chronically infected follicles require the same treatment as given under the complications of acute anterior urethritis. Chronic prostatitis and chronic seminal vesiculitis, unless of very long standing, will be much benefited by hot rectal irrigations. In addition, they call for massage by means of the finger introduced into the rectum. The irrigations may be practised every day at first and later every three or four days, according to indications. The digital massage, however, should not be repeated oftener than once in from five to seven days, and should be discontinued altogether for a time if it aggravate rather than relieve the conditions.

ON THE USE OF THE FIXED CATHETER IN THE TREATMENT OF URINARY INFECTION AND OF PROSTATIC AND URETHRAL HEMORRHAGE.

CLINICAL LECTURE DELIVERED AT NECKER HOSPITAL.

BY FÉLIX GUYON, M.D.,

Professor of Surgery of the Urinary Tract at the Paris Faculty of Medicine.

Gentlemen,—I have just called your attention to a case in our ward in which the fixed catheter was used on several occasions; the patient bore it for forty-nine days, and it put an end to an entire series of troubles from which he was suffering. On the 31st of last month he was admitted to the hospital in a pyretic state (38.5° C.), suffering from a chronic cystitis that had again become very acute, accompanied by intense posterior pericystitis, and urinating every few minutes to empty a bladder which had entirely lost its holding capacity and would not tolerate the introduction of the smallest amount of liquid. He was at once treated by the fixed catheter and by instillations of silver nitrate. His fever disappeared on the second day, and we were able not long afterwards to examine the interior of the bladder, in which were found a number of phosphatic calculi. These were crushed two weeks after his entrance into the ward, the result being verified five days later, when the bladder was found entirely free of calculi.

These two applications of instruments appeared at first not to be followed by a rise of temperature; but later a series of pyretic manifestations, each lasting a day or two, occurred, and did not cease until quite recently. In every instance the rise of temperature was due to imperfect working of the fixed catheter, and a change in the catheter immediately put a stop to the trouble.

On account of the repetition of these pyretic manifestations and of the marked condition of pericystitis, I several times suggested to the patient that cystostomy would be of benefit to him. This operation he refused. To-day his feverish turns have com-

pletely stopped, he empties his bladder satisfactorily, the pericystitis has almost entirely disappeared, and his general condition has become quite normal.

I propose to-day to take as my text this man's history, which is interesting because of the complexity and gravity of the disorder, the very long period during which urethro-vesical drainage was continued, the mishaps which disturbed the otherwise satisfactory way in which the fixed catheter was tolerated, and the final result; and shall, with the help of the cases that are constantly seen in our wards, describe the treatment of infection of the urinary tract by means of the fixed catheter.

Some cases of urethral and prostatic hemorrhage having recently occurred in our wards, I shall also make a few remarks on the treatment of this symptom by the same surgical means. The employment of the fixed catheter is the method of vesical drainage to which preference is given in the treatment of febrile urinary infection. It is resorted to when the general indications of infection do not yield to the sufficiently repeated use of the catheter and to methodical bladder-washing. Under such circumstances it is evident that the bladder and kidneys are affected and that the patient's condition is serious or will soon become so.

As regards the condition of the kidneys, there are certain cases to which particular attention should be directed. In some patients the volume of one of these organs will be found noticeably increased, but will gradually diminish during the course of the treatment.

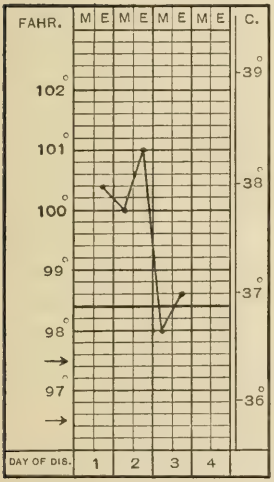
I have for a long time observed that in serious cases of urinary infection urethro-vesical drainage by means of the fixed catheter gives results that are remarkable both for their rapidity and duration. In 1895 I examined the histories of forty-nine such cases, and proved this statement by showing that thirty-three recovered, making twenty-three per cent. of failures, which were not followed by death. To-day mention may be made of fifty additional cases, with forty-two recoveries and eight deaths (sixteen per cent. mortality).

When treating the subject in 1895 I compared the results of this method with those given by suprapubic drainage, which at that time showed thirty-five per cent. of deaths. To-day, if the new set of cases is confronted with the figures given in Poncet and Delore's recent treatise on cystostomy, it will be found that the latter opera-

tion shows thirty-six and two-tenths per cent. of deaths, whereas our method in the later cases does not exceed sixteen per cent.

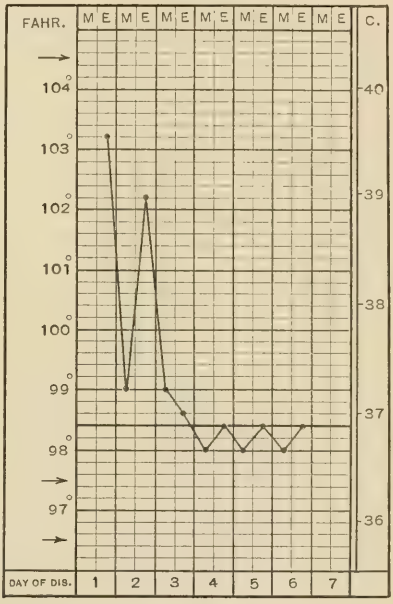
The results obtained by the fixed catheter continue to be distinctly better, and the above figures prove the curative action of drainage by the natural passages in febrile urinary infection; but there are facts even more conclusive,—the results furnished by the study of the modifications produced on temperature by the fixed catheter. The conditions under which they are observed are always the same; the same circumstances precede and accompany the in-

FIG. 1.



Rapid fall.

FIG. 2.



Rapid fall.

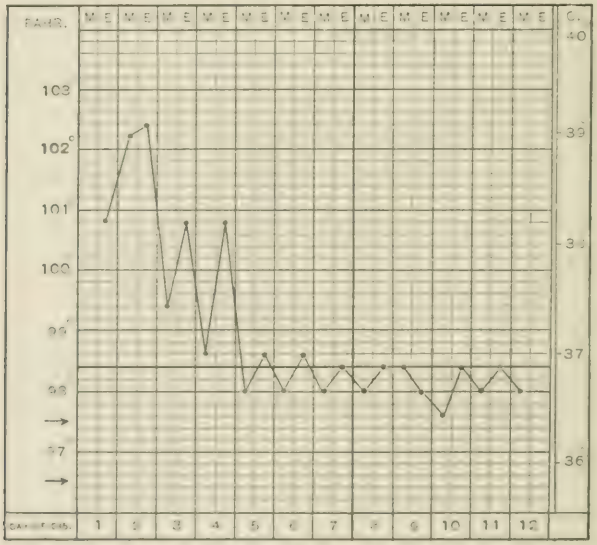
crease and decrease of the abnormal temperature, and we can settle with experimental precision the relations existing between the phenomena and their causes.

The time requisite to bring a high temperature to the normal by means of the fixed catheter in febrile urinary cases varies somewhat in different patients. Thus, in our cases of 1895, apyrexia was obtained most frequently on the third day (thirty-four per cent.), next on the first (twenty-four per cent.), and finally on the second (eighteen per cent.). The present set of cases shows slightly

different results; but this difference (which is apparent only) can readily be explained. In the former set apyrexia was placed after the last great oscillation of temperature; in the present set we take into consideration all the small subsequent oscillations, and continue until the temperature becomes normal in the evening. In the former case apyrexia is not established, but is indicated, as it were; in the latter it is actually obtained. The latter method is more rigorously exact and better adapted to the requirements of practice.

Proceeding in this manner, the figures obtained are as follows:

Fig. 3.



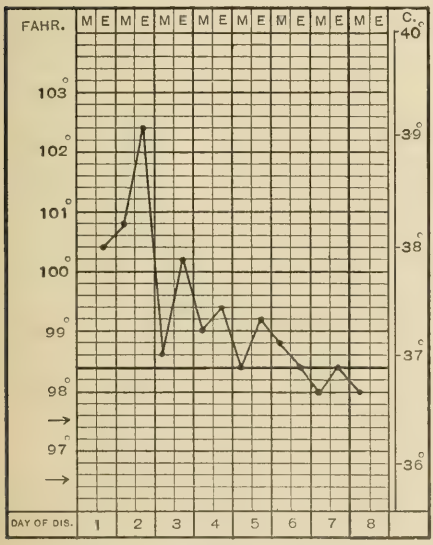
Progressive fall.

complete apyrexia occurred eleven times on the second day (thirty-four per cent.), eight times on the fourth (twenty-four and four-tenths per cent.), and seven times on the fifth (nineteen and six-tenths per cent.); in cases of serious infection seven days were required in one case and nine in another; fifteen times out of forty-two a normal temperature was obtained inside of forty-eight hours, and in thirteen other cases within four days. The initial temperature often reached 39° C. and sometimes 40° C. (104° F.).

The manner in which the fever descends varies: (1) rapid descent in from twelve to thirty-six hours (see Figs. 1 and 2);

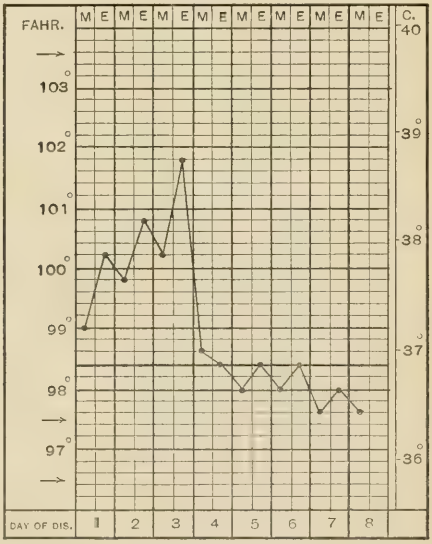
(2) gradual descent, of which Fig. 3 is an example. This second method occurs in chronic forms of infection, or in recurrences caused, for instance, by premature removal of the fixed catheter; in such instances six, seven, and even nine days elapse before the temperature is normal in the evening. But while progressing slowly in these cases, the effect of the fixed catheter is usually immediate, though with a few patients it appears at first to have no effect whatever on the course of the temperature, which continues to rise although the catheter is working perfectly; still, after twenty-four hours (and this period is practically never exceeded)

FIG. 4.



Gradual fall of temperature.

FIG. 5.



Gradual rise of temperature with rapid fall.

the descent occurs, and is rapid or slow according to the degree of infection. (See Figs. 4 and 5.) Cases have been recorded in which the temperature was not at all affected by the fixed catheter, and continued to rise. These, however, are very unusual. Even in the most serious forms of urinary infection the fixed catheter often produces a fall of temperature, though the latter may be but momentary and followed by a steady rise. In such cases danger to life is extreme. Drainage by the natural passages, when it does not prevent the continuation of the rise in temperature, even though

it causes a temporary fall, is of no use. In like conditions hypogastric drainage has also failed in our hands.

The patient to whom chart No. 6 belonged was a urinary case with serious infection. The fixed catheter lowered the temperature temporarily, the fall from evening to the following morning being from 39.4° to 37.2° C.; but the patient succumbed two days later, and the autopsy revealed bilateral suppurative nephritis. In this case the ultimate failure is easy to understand, but it is impossible not to be struck by the immediate result produced by the use of the fixed catheter.

The above facts show clearly the effect of vesical drainage on the fever symptom, while those described below prove that the fall in temperature is due to this measure alone. Fever reappears when the catheter is removed too soon or when it does not work properly. Thus, to the proof already given by the results obtained by means of the fixed catheter is added the counter-proof supplied by removing or hindering the drainage. To know when drainage may be discontinued without exposing the patient to a recurrence of fever is a delicate and very important point of practice, the more so when it is remembered that recurrence takes place at once, and in some instances makes a more prolonged treatment necessary. We should be guided by the following rules:

Complete cessation of all rise of temperature, however insignificant is the necessary condition. As has already been said, one cannot consider the fever over until the evening temperature no longer exceeds 37° C.

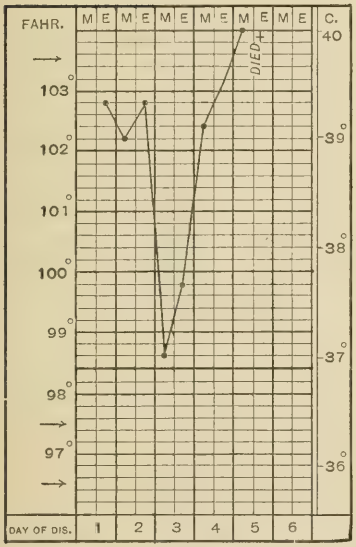
Before deciding on removing the catheter the temperature markings should be identical for several days. Two or three days are enough when the fall in temperature is rapid and unmistakable, with no period of small oscillations. In other cases it is prudent to watch for five, six, or seven days, according to the time required for the temperature to fall and the manner in which it falls, and it is well also to take into consideration the decrease or continuation of the other signs of infection, such as the condition of the tongue and lungs and the existence of somnolence.

A strict observance of these rules will protect us from failures that are to be attributed, not to the fixed catheter method, but to the way in which it is applied. Success will be insured by not removing the fixed catheter prematurely, and at the same time the

patient will not be subjected to an unnecessarily prolonged treatment.

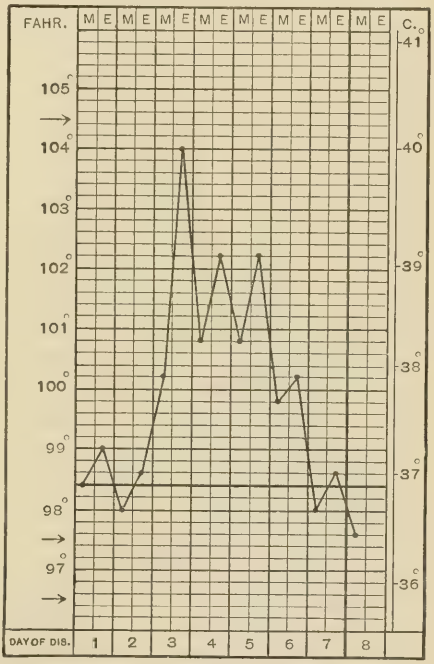
If the length of time necessary to obtain defervescence be added to that required to be certain that it has been obtained, it will be found that most patients keep the fixed catheter less than a week,—generally four or five days; and that those most seriously affected keep it sometimes for two weeks, rarely more. The length of the treatment of the accidents of urinary infection by the fixed catheter

FIG. 6.



Temporary fall of temperature followed by rise again, and death.

FIG. 7.



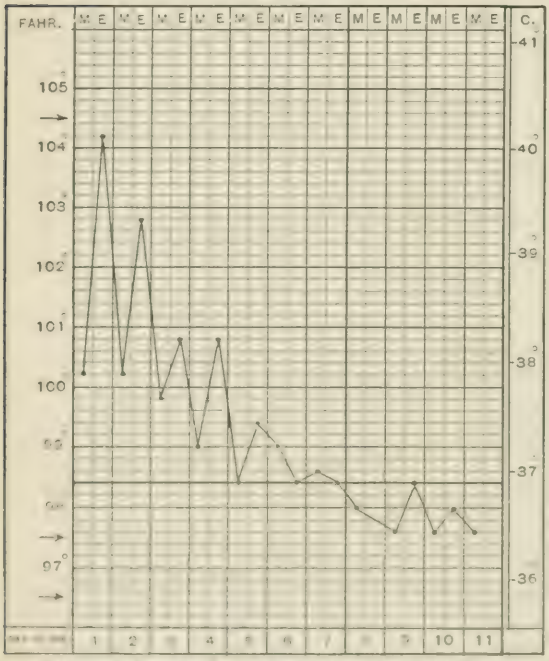
Catheter removed too soon by the surgeon.

is therefore not great, and I insist on this point, as the length of the treatment is a favorite argument with those who oppose the method. In any case, this mode of treatment can be prolonged without inconvenience; the patient mentioned at the beginning of this lecture kept up this treatment for *seven weeks,* and begged us not to withdraw the catheter, as whenever this was done his fever returned. I mentioned a similar case in an article published in conjunction with M. Michon. With the patient in question cys-

totomy gave a good result. This operation is indicated when the effect of drainage by the natural passages, although relatively favorable, is insufficient in spite of its methodical use. Cases of this sort are very uncommon, if we are to be guided by our hospital experience.

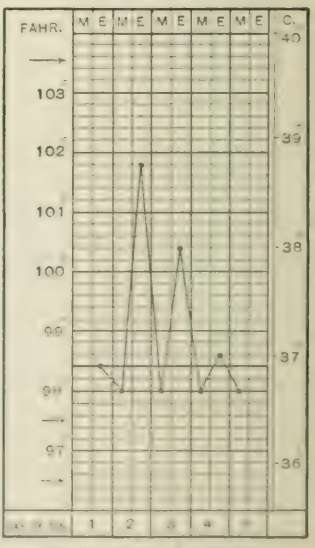
Fig. 7 shows you the result of premature withdrawal of the catheter. On the following morning the temperature rose to 38° C., and the catheter was replaced. In spite of this the temperature continued to rise and reached 40° C. by evening. But on the following morning it began to fall, and was normal again on the fourth day.

Fig. 8.



Catheter taken out by the patient on the eighth day.

Fig. 9.

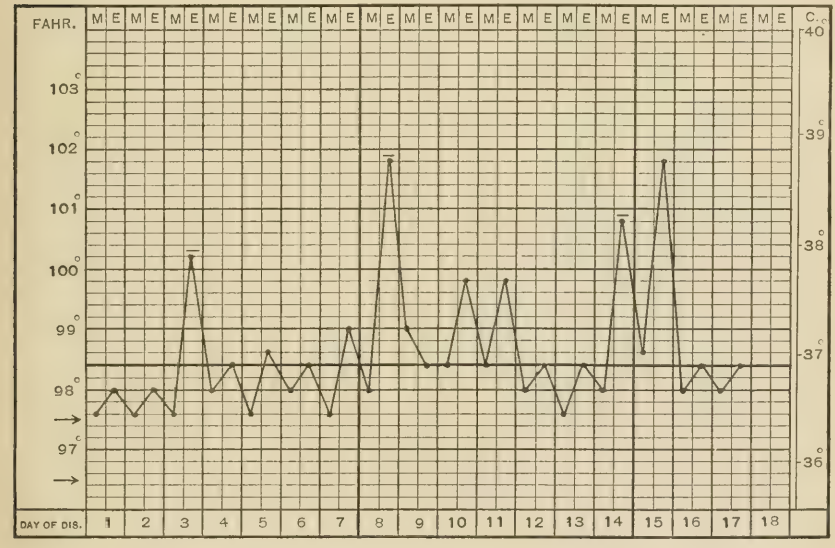


Displacement of catheter.

Fig. 8 is an example of the same sort. The catheter was surreptitiously withdrawn by the patient during the night. His temperature had been normal for only one day after six days' treatment. The catheter was replaced in the morning, but by evening the temperature reached 40° C. The next morning it began to descend, and finally became permanently normal.

An immediate and more or less marked rise in temperature shows that the catheter has been withdrawn too soon. This rise may be accompanied by rigors and by reaction on the general state of health. Whatever may be the degree and importance of the symptoms that accompany the rise, the indication is clear: the fixed catheter must at once be reinstated. There is all the more reason for following this rule when one sees patients who, having quickly and completely got rid of their fever in one or two days, accomplish the same end a second time, after prematurely removing their catheters, only after three or four days. You must therefore not discontinue urethro-vesical drainage too soon; the rules laid down afford all the security desirable without extending the treatment unduly.

Fig. 10.

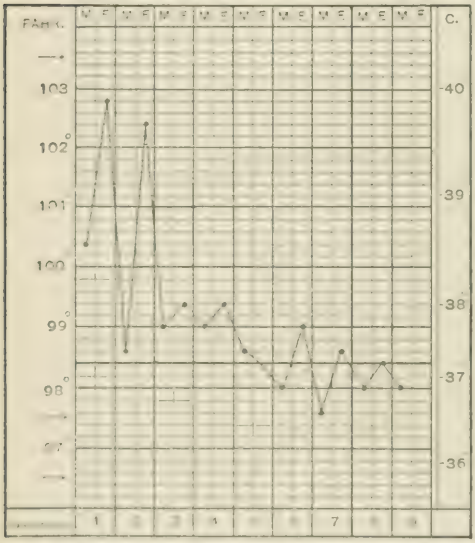


Repeated obstruction of the catheter.

When the fixed catheter gets out of place or becomes obstructed, fever will either return or fail to disappear when still present. The accidents that occur under these circumstances are less serious and more easily counteracted, as drainage has only been hindered, not suppressed, or at any rate for a short time only. In the majority of cases a change in the position of the catheter quickly improves the situation.

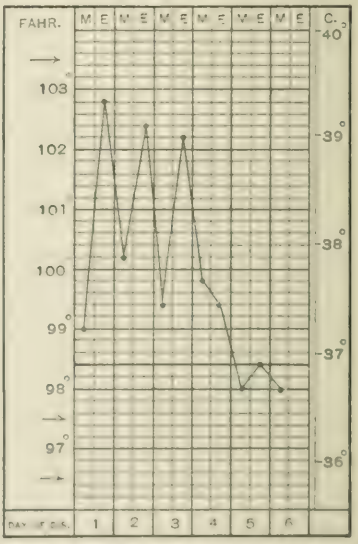
In Figs. 9 and 10 will be seen the consequences of the catheter getting out of position or becoming obstructed. No. 10 represents a portion of the seven weeks' chart belonging to the patient cited at the beginning of this lecture. In the case of this patient the urine clogged with unusual facility and damaged the catheter. Such cases are rare, but one must be prepared to meet them. Frequent changing of catheters is then particularly indicated. As in all forms of drainage, here also the cleansing and replacing of the tube are the surgical conditions to be fulfilled if the process is to be efficacious.

FIG. 11.



Vesical infection with increase in size of one of the kidneys.

FIG. 12.



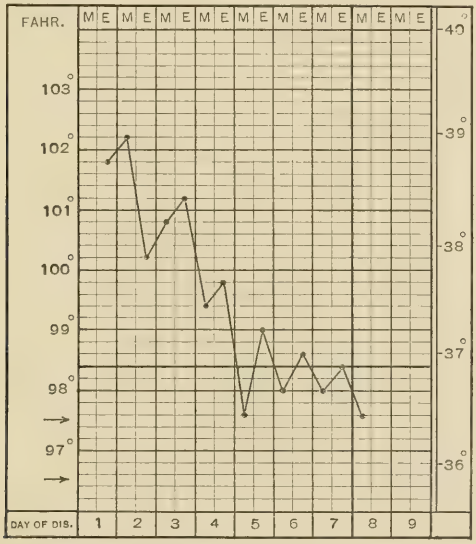
Vesical infection with increase in size of one of the kidneys.

I think it advisable to also call attention to Figs. 11, 12, and 13, belonging to prostatic patients, with whom may be noticed, among the general accidents of urinary infection, a very marked increase in volume of one kidney. As will be observed, in these cases also the temperature fell when the fixed catheter was installed; the fall, however, was not very rapid, and a few wide oscillations preceded the small ones, which continued for some time. But in the case which lasted the longest (No. 11) defervescence was complete on the sixth day.

In these complex cases the fixed catheter also brings the temperature down; this occurs in the usual conditions more slowly, but with the same progressive fall, without that steady rise the gravity of which is known.

A still more noticeable point is that under the influence of the fixed catheter the enlarged kidney decreases in size. In the three cases just referred to, progressive diminution of the kidney followed the application of the fixed catheter, and the enlargement has not reappeared. Are we to infer from this that

FIG. 13.



Vesical infection with increase in the size of one of the kidneys.

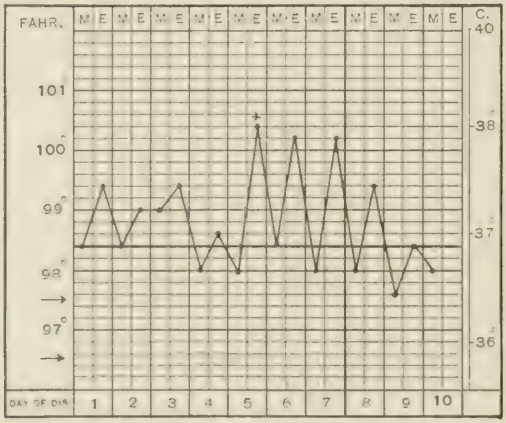
the fixed catheter causes the evacuation of matter retained by the kidneys, or does it put an end to a state of renal congestion? This cannot be decided as yet; the main point to be remembered is that increased renal volume is not a contraindication to the use of the fixed catheter in urinary infection. However, I may add that I have observed several cases of renal retention definitely cured under the treatment of vesical retention only.

All the cases that we have been analyzing demonstrate the same theme. They bring to light the conditions under which pyrexia disappears and reoccurs, and they prove one another. The way

in which they occur is thus surely established, and the conclusions to be drawn cannot be disputed.

Drainage *per vias naturales,* when properly conducted and watched, brings down pyrexia. Defervescence is rapid or progressive; once obtained, the temperature should not rise again. Delay in bringing about apyrexia, as well as recurrence of pyrexia, is due either to the catheter not working properly or to its premature withdrawal; these accidents end when it is replaced or its position is changed. Therefore, when these conditions are properly filled, and apyrexia does not occur, and particularly when the fever does not decrease, or even increases, although the catheter

FIG. 14.



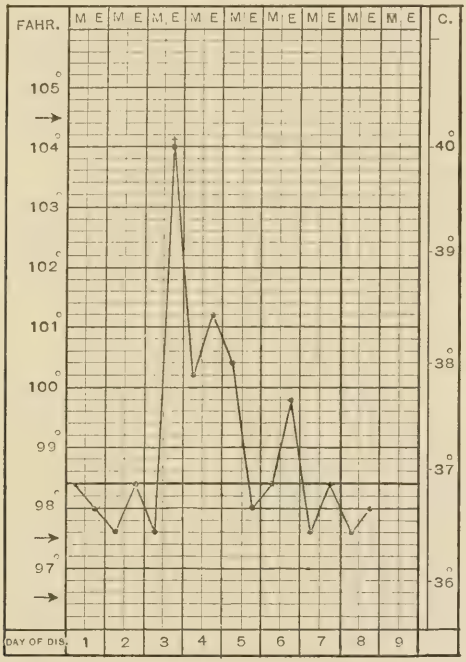
Prostatic abscess.

is working satisfactorily,—before coming to the conclusion that the failure of the fixed catheter to produce its usual result is due to the gravity of the infection of the urinary tract, look carefully for some other infectious localization. Examine the genital apparatus, prostate, testicles, seminal vesicles, etc. Auscultate the lungs frequently, without being too prone to admit the influence of pulmonary congestion, which is usually only a consequence of the urinary infection; treat it medically in the usual way, but a good urinary drainage is the most efficacious remedy. This, however, is not the case with other forms of genital infection, which must be attacked in a direct manner, locally, when diagnosed. They should be opened up as early as possible,—almost prematurely,—and in no

case more so than in affections of the vaginalis; of all the genital foci this is the one that should be incised the earliest.

Patients who have been seriously infected for long periods are very often subject to secondary genital infection; but this is very rarely observed in patients treated by the fixed catheter, and it can readily be demonstrated from our list of cases that this method does not favor its occurrence. In any event, it in no way hinders its cure. Fig. 14 represents a patient with whom defervescence

Fig. 15.



Orchitis and vaginalitis.

was incomplete, and whose temperature afterwards rose again for four days, although the catheter was working properly. A small prostatic abscess then opened into the urethra, and on the fifth day apyrexia was complete and remained so. Fig. 15 is that of a patient whose fever appeared to have subsided entirely and whose catheter I was about to remove, when his temperature suddenly rose to 40° C., and took four days to return to the normal; the appearance of orchitis on the right side, which retrograded rapidly

although the catheter was kept in place, afforded an explanation of the rise in temperature. The treatment of urinary infection by the fixed catheter must therefore be continued in spite of the appearance of genital foci.

Infectious genital localizations, whether primary or secondary, are not contraindications to the employment of the fixed catheter, and this is also true of renal localizations of the importance of those we have mentioned; they do not prevent its use in any way. Drainage by the natural passages has a remarkable curative effect on the latter and does not hinder the recovery of the former. Clinical experience, therefore, warrants us in considering the fixed catheter method as the preferable one in the treatment of urinary infection.

The ulterior results of the method—which can be observed at once, as the patients show them as soon as the catheter is removed—confirm this conclusion. In our wards patients may every day be seen who have been under the fixed catheter treatment for serious infectious accidents, and who have remained cured after it was removed. Provided they empty the bladder by regular catheterizing, and wash it out regularly, they find themselves in the usual position of prostatic patients with retention.

I have still to speak about the action of the fixed catheter on prostatic and urethral hemorrhage. Whatever may be its origin, whether due to congestion, neoplasm, or traumatism, whether or not the blood has accumulated in the bladder and we are obliged to aspirate the clots, prostatic hemorrhage yields rapidly to the fixed catheter. Two patients in our wards have shown this quite recently. The first one had a very large prostate and a calculus four centimetres in diameter; exploration with a metallic sound produced a serious hemorrhage which brought him to the hospital as an urgency case. The fixed catheter, passed into the bladder over a metallic guide, put an end to the flow of blood in forty-eight hours; it was then left in place for a week so as to modify the canal, after which a crushing operation was performed with no loss of blood. The second patient is still in the hospital, to which he was admitted for prostatic hemorrhage produced by catheterizing; in three days the fixed catheter put a stop to this accident. In both of these instances the result was excellent and quick.

Traumatic urethral hemorrhage due to the unskilful use of metallic sounds also yields rapidly to the fixed catheter. Two such

cases with serious hemorrhage have recently passed through our wards. In forty-eight hours the flow had ceased, two days later the fixed catheter was removed, and the patients had then only to wait for a suitable time to have their urethral disorder treated properly.

These various facts demonstrate that the fixed catheter is a first-class therapeutic means which, in addition to its marked efficacy, has the advantage of telling the surgeon at once when it is not working properly. It must, however, be carefully applied and closely watched. Are there drawbacks to its use? Those to whom is confided the treatment of prostatic patients in serious attacks of urinary infection will not think so, I am sure.

THE USE OF MERCURY IN THE SYSTEMIC TREATMENT OF SYPHILIS.

THREE LECTURES DELIVERED TO THE MEDICAL STAFF OF THE ST. LOUIS CITY HOSPITAL.

BY A. H. OHMANN-DUMESNIL, M.D.,

Consulting Dermatologist and Syphilologist to the St. Louis Health Department.

I.

GENTLEMEN,—Before entering into a consideration of the subject before us, I desire to state that it is not my purpose to take up the discussion of the local use of mercury in syphilitic lesions. What I propose to speak of on this occasion is what is known in ordinary practice as the constitutional treatment of the disease by means of mercury. Mercury is here taken in its broad sense, and it is this very indefiniteness which has proved such a stumbling-block in the path of those not well acquainted with the therapeutics of syphilis. I do not propose to discuss in detail the relative advantages of mercury and iodine, for, though each has its proper place and time for administration, syphilis may be successfully treated without iodine, but not without mercury. So far as mercury is concerned, I also wish to speak of the different forms in which it is employed, the advantages and disadvantages of each, in what syphilitic conditions certain preparations are most advantageous, and, finally, how and in what doses they should be administered.

If we are to believe the researches of certain historiographers, the use of mercury in syphilis is very ancient. Some contend that cinnabar was employed by the Chinese thousands of years before the Christian era. This is doubtless an exaggeration; but that it was used several centuries anterior to the advent of Christ is proved by documentary evidence. We learn from Roman writers that physicians did not treat venereal diseases, but courtesans, midwives, and charlatans lined the roads leading to the temples of Venus and offered their ointments for sale. These ointments were for the cure of syphilis, and that they did cure was evidenced

by the votive pictures and models which were hung on the walls of the temples. It is supposed that the preparations were mercurial in character. But perhaps the best evidence of the use of mercury at an early period is recorded by Rabelais, who speaks of a certain Pope, "a rubber in of the mercurial ointment for the poxy." I will not enlarge on the barbarous methods in vogue in the Middle Ages, such as applying mercurial ointment with wooden paddles, and placing patients in ovens heated to such a degree that more were taken out dead than alive, but will simply consider the more modern efficient methods.

The different modes of treating syphilis with mercury may be divided as follows:

1. Fumigations.
2. Baths.
3. Inunctions.
4. Internal administration of liquids.
5. Internal administration of solids.
6. Hypodermic injections of insoluble salts.
7. Hypodermic injections of soluble salts.
8. Intramuscular injections.
9. Intravenous injections.

Of course, each method has numerous modifications, and many different preparations are employed to bring about the desired result. There should also be taken into consideration the facts that some have a very rapid transitory effect, and that the action of none is permanent. Despite the claims which have been made by many, it is a fact that, as in the case of knowledge, there is no royal road to success. It is persistent treatment alone that ultimately cures syphilis, and no arbitrary rule can be laid down as to the length of time it will require. Patients apparently cured, and rejoicing in the idea, ofttimes suddenly awaken to the tortures of parasyphilitic troubles, followed by deep syphilitic affections or so-called tertiary lesions of a destructive nature. All of these are doubtless due to insufficient treatment at a time when a proper course would probably have prevented their occurrence. It is on this account, I repeat, that much caution must be used in formulating a prognosis, and the obedience and conscientiousness of the patient are prominent factors in the consideration of the question.

If we are to believe in the authenticity of certain Chinese documents, venereal diseases were known to them several thousand years before the Christian era. Not only this, but they recognized the chancre, certain local syphilitic manifestations, and the generalized eruption. We are also told that the Chinese idea of the therapeutics of syphilis, though a good one, was somewhat restricted, being confined to fumigations with cinnabar,—a treatment which was not adopted in Europe until within a comparatively recent date, and never very enthusiastically received, on account of the slowness of its action and the inconvenience of its application. That mercury acts upon the entire organism is very plainly evidenced by the fact that mercurialism is produced. There are ptyalism, swelling of the buccal mucous membrane, tenderness of the gums, loosening of the teeth, diarrhœa, etc. But many fumigations are required before this degree of saturation is attained, and for this reason the method is not usually employed. However, as you might be tempted to undertake it, I shall briefly describe the treatment. In the first place, the patient should be prepared for the fumigation by taking a hot bath and being well rubbed down. He is then seated on a chair having large perforations in the seat. Underneath the chair is placed an apparatus which is intended to volatilize the mercurial preparation employed. This volatilizer consists of an alcohol lamp, over which, supported by a tripod, is an iron saucer having a capsule in the centre to contain the mercurial and a circular gutter at the circumference in which water is poured. The patient being seated, a blanket is attached about him in such a manner that it will fall to the floor and completely enclose both him and the chair. The mercurial having been placed in the little cup, the lamp is lighted, the blanket well closed, and the fumigation begun. When the mercurial has been completely volatilized, the patient is well wrapped in the blanket and put to bed. In a half-hour he may be permitted to resume his clothing, and on the next day is ready for another fumigation. With the exception of some slight details, the method above described is the same as that advocated by others.

An important question which naturally arises is as to what preparations are used and in what doses. The two principal salts of mercury employed for fumigations are the heavy ones, the protochloride and the sulphide; in other words, calomel and cinnabar.

The amount of calomel necessary for one fumigation is about forty grains, of cinnabar about eighty grains. Experience teaches that a mixture of the two in the proportion of twenty grains of calomel and forty of cinnabar, is much more efficient than either one used alone. The heat should not be too great, as it will cause the patient to perspire so freely that the mercurial fumes will rapidly be condensed and washed off instead of being deposited in an impalpable powder upon the surface of the body.

Whilst this method has its advantages in the treatment of the local manifestations of syphilis, more especially in the earlier periods, it is much inferior to others that will be mentioned as we progress in the consideration of the different modes in which mercury is administered. Even when applied locally, the remedy is much inferior to others, the method somewhat complicated, and the results rather tardy. It might be of advantage in the systemic treatment of the disease could the fumes be inhaled; but, unfortunately, this is not possible, and consequently the only method in which fumigations might be employed with advantage must be discarded, and the treatment looked upon as a therapeutic curiosity rather than one of real worth.

So far as the employment of baths is concerned, the method seems to be almost entirely restricted to cases of so-called hereditary syphilis in infants and very young children. In such cases the action of the baths is very beneficial. They are indicated more particularly in those severe forms of syphilis in which a marked impression must be made upon the disease. Thus, when bullous manifestations are present, or there are bone-lesions, or icterus and spleen-trouble, no hesitation need be felt in using them. Care, however, should always be exercised in the employment of mercurial baths, as they bring on ontoward symptoms in some cases. The mercurial to employ is corrosive sublimate. From seven to twenty grains of the bichloride are dissolved in a tumbler of hot water by the aid of an equal quantity of muriate of ammonium. This is then put in a bath of seven or eight gallons of water, and the child permitted to remain in it from five to eight minutes. At the beginning of the treatment baths should be given daily, then bi-weekly, and this is kept up until from twelve to thirty baths have been taken. All untoward symptoms should be watched for, and the moment they manifest themselves the treatment should be dropped.

A method of using mercury in the treatment of syphilis which was in vogue in the Middle Ages, and which is to-day very popular and regarded by some syphilologists as the best, is that by inunctions. You have seen me obtain most rapid results in this hospital by its use, and the great reputations of the hot springs of Arkansas and of Aix-la-Chapelle are almost solely based upon this method. It is largely employed in Germany, Austria, and Russia, but to a less degree in the other countries of the Continent, Great Britain, and the United States. However much it is lauded, no matter what wonderful results are procured by it, my advice to you is always to watch your case. It is here that you will find it profitable to keep in mind the old Roman maxim, *Caveat emptor.*

The form of ointment to be used for friction is one of the questions to be considered, as well as the quantity and frequency of applications and the method to be observed. In the first place, you will find a large number of mercurial ointments recommended, each one being lauded by its particular advocate, and some of which are very difficult to obtain or to prepare even when the formula is at hand. I have found the unguentum hydrargyri very satisfactory. Not the mercurial ointment of the United States Pharmacopœia, which contains oleate of mercury, nor the ointment made with vaseline, but the old-fashioned unguentum hydrargyri cinereum, the Neapolitan ointment, the gray ointment of the French, or the well-known blue ointment. This latter should contain fifty per cent. of metallic mercury which has been rubbed up with lard until complete extinction of the globules, not under a microscope, but under a good magnifying-glass. There are some syphilologists who prefer making a mixture of mercury, lanolin, water, and tincture of benzoin, but they hardly present better results than do others with the ointment which you have seen me use.

The next question which presents itself is in regard to the quantity that should be employed for each inunction. In the case of an adult sixty grains are considered sufficient. But, without explanation, this is misleading. My rule has been to order for those patients who remain in the city or in a public hospital a quantity whose bulk is the same as that of the distal phalanx of the little finger. For those who are at hot thermal resorts much larger quantities are not only given, but are necessary for the simple reason that the ointment is being continually washed off, and necessarily

not as much is absorbed. This is no doubt the reason why such patients can safely support more of the ointment. As regards the frequency with which these inunctions should be made, their uniform daily application until it is judged that a sufficient number have been taken is now pretty generally accepted. This method is subject to modification, however. At thermal resorts no days of intermission are permitted to occur, whilst in urban and hospital practice they are. Thus, as you have seen the applications made here, the patient rubs in the ointment upon the inner surface of the thighs one day after another, then the inner surface of each arm is "rubbed" upon successive days, and, finally, the sides of the chest. This takes six days. The directions are to rub in the ointment until it is no longer perceptible. On the seventh day the patient takes a hot bath and cleans the skin of the accumulated ointment and epidermal scales. This is a day of rest. The inunctions are then resumed in the same order until it is judged proper to suspend them. The time to do this is not to be determined by the disappearance of symptoms, but rather by the signs indicative of the marked action of the mercury. Care must be taken to keep the teeth clean, the mucosa of the mouth in good condition, and, in fact, all those precautions must be observed which are necessary to prevent the local symptoms of mercurialism. Directly any of these show themselves the treatment must be suspended.

The effects of inunctions of mercury in syphilis are both marked and rapid; but, unfortunately, they are not permanent. If you will recall that eleven-year-old girl in Division 2, who came into the hospital with the chancre still apparent, some few papules, and later marked condylomata of the genitals, you will remember that all these symptoms had almost entirely disappeared after four inunctions. This was certainly encouraging; but, even after a thorough course of frictions with mercurial ointment she will have to be treated internally with other preparations of mercury. Speaking of these rapid good results from inunctions recalls a case to my mind. A gentleman who had contracted syphilis went to Hot Springs to be treated. He underwent a thorough course of mercurial inunctions and was pronounced cured of his disease. When I saw him twelve years had elapsed, and he was suffering from a relapse. In a very short time he was relieved of the symptoms. Fortunately, they were cutaneous. However, when the relapse

manifests itself in the form of parasyphilitic symptoms a much more serious condition presents itself, and one which but too frequently is accompanied by organic changes.

For these reasons you had best be cautious in making promises in cases of syphilis that are treated by mercurial inunctions. The possibility, or rather certainty, of a relapse should be insisted upon, and the patient fully apprised of what to expect. After a certain period of rest, which should not be permitted to extend for too long a time, mercurial treatment should again be pursued. Inunctions will be of advantage, but other methods will also be found good. Whatever the course you adopt, always tell the patient the truth, warning him of the dangers of neglect, and by following such a course you will find your results of the best.

II.

Before entering into a consideration of the internal use of mercurial preparations it may not be uninteresting to mention the fact that attempts have been made to treat syphilis by the application of mercurial plasters, in which, by incorporating the metal with a base containing rubber, it was supposed that the skin would be softened and thus a more ready absorption of mercury promoted. This was a mistaken idea, however, and the results hardly justify such a clumsy and disagreeable device. Indeed, it very soon became apparent to the upholders of this method of medication that it was more properly adapted to the local than to the systemic treatment of syphilis. In order to obviate the disagreeable features of mercurial inunctions the mercurial vest was introduced. It consisted of a chamois undergarment very similar to a chest protector, into which mercurial ointment had been rubbed. This clumsy device was never satisfactory, as there was no exact gauge as to the amount of mercury used, and it soon fell into desuetude. It, however, has been followed by a late device known as mercolint. This is also an undergarment, but one which is accurately dosed. There are three strengths, and the quantity to each cubic centimetre is given. It may be worn until the composite cloth which holds the mercury becomes perfectly white. When this occurs the amount of mercury absorbed is exactly determined by the physician. This method is rather popular in Germany, but has not yet obtained a firm hold in this country.

What I desire to call your attention to to-day is the internal administration of mercurials in solution and in solid form. The liquid forms of mercurial preparations are far less numerous than the solid; in fact, there is but a very small choice, and in this we must be largely guided by the conditions presented, by the facility which is afforded for their administration, and by the peculiar idiosyncrasies of the patients. You will find that all of these are factors of no small importance. There is another question which must never be lost sight of: it is that of the time factor. There must always be taken into consideration whether the continuous or the intermittent plan of treatment should be followed. In other words, whether full or tonic doses should be administered to a syphilitic. This question of dosage largely depends upon the ability of the physician to see the patient frequently or at long intervals only. As you can readily see, this is an important matter in private practice,—more so than in hospitals, in which patients are under constant surveillance, and not allowed to neglect any detail, even if they are inclined to do so.

The most prominent and one of the most useful mercurials to administer internally in soluble form is corrosive sublimate or the bichloride of mercury. It is necessary here to obtain a perfect solution and to see that it is sufficiently diluted, so as not to bring on any gastric irritation. For two reasons I have always been opposed to the making of a solution by the aid of alcohol. In the first place, it is bad practice to administer alcohol in any form to a syphilitic; and, secondly, it does not perfectly dissolve corrosive sublimate. In even a moderate-sized bottle some undissolved bichloride will in a few days be found at the bottom, which has two bad effects: the uniform strength of the solution is destroyed, and that portion near the bottom is very irritating. To obviate these disadvantages I have been in the habit of using muriate of ammonia as a solvent, in a proportion equal to twice the amount of bichloride. You may remember that the ward mixture I have ordered is made as follows:

℞ Hydrarg. bichlorid., gr. xvi;
Ammon. muriat., gr. xxxii;
Aquæ destillat., f℥xvi.—M.
Sig.—A teaspoonful in water after each meal.

As you can readily see, this makes an eighth of a grain at a

dose, and this is a full dose. Of course, it is too much for some patients, but they are few. I have more often found it necessary to increase it than the reverse. There have been cases in which it was necessary to increase the dose to such an extent that one grain was administered thrice daily, and not until that had been done did the patients improve. To be candid, I never could understand how doses of one-twentieth or one-sixteenth of a grain of this remedy could ever be expected to produce any satisfactory results. The disease cannot be trifled with, and it establishes an amount of resistance to mercurials which the physiological individual would never exhibit. All physiological experiments to establish mercurial tolerance are misleading. The premises being false, the conclusions must necessarily be erroneous. Such doses are adapted to an intermittent plan of treatment only. But, on the other hand, smaller doses cannot be used continuously, as salivation will easily be induced. You will find, however, that solutions of corrosive sublimate are as useful in the systemic treatment of syphilis when taken internally as they are for antiseptic purposes in operative surgery. In fact, the one remedy for the treatment of lues is bichloride of mercury. It is without doubt the superior of all others, and produces results with almost mathematical exactness.

So far as the soluble preparations of mercury in the treatment of syphilis are concerned there is small choice. A preparation which has enjoyed some popularity of late is an alterative rather than an antisyphilitic. It was devised by Dr. Barclay, and is a bromide of mercury, arsenic, and gold known under the name of *mercauro*. It is well spoken of by many reliable gentlemen, so that we must needs accept their conclusions. It seems to be most efficient in the parasyphilitic phenomena. If you will consider for a moment that it contains arsenic, you will readily understand why the efficiency of the small dose is increased. It is a well-known fact that arsenic is synergistic to mercury, and this is often taken advantage of in treatment. It prevents the administration of toxic doses of mercury, and permits of the attainment of results which otherwise could not be expected.

So far as the administration of solid mercurial preparations in the treatment of syphilis is concerned, there is *un embarras de richesses*. They may roughly be divided into two classes, powders and pills. The salts susceptible of being made up in these forms

are almost as numerous as are those employed in hypodermic injections, of which more anon. The simplest mercurial powder is calomel. This, as you know, is the mild protochloride of mercury, than which—with the possible exception of quinine—no remedy is more generally used; and yet it is probably least frequently employed in the treatment of syphilis. It is not unpleasant to take, but is not popular with patients on account of its effect on the bowels. It easily induces diarrhœa; and whilst some practitioners endeavor to correct this untoward effect by combining powdered opium with it, they merely lay the foundation for a habit which is worse than the disease for which the calomel is administered. The unfortunate creature who is placed under such treatment is made miserable if opium is persisted in, and becomes, in time, an irreclaimable opium "fiend." Calomel will not produce good results unless given in grain doses at least thrice daily, and even then it is a question, in my mind, whether they are as good as could be obtained with other preparations. I cannot speak from experience on this point, as I never liked the method and hardly cared to enter upon any experiment of doubtful value. It is profitable neither to the patient nor to the physician. However, another powder that is employed, and with which every one will be satisfied, is mercury with chalk (gray powder, or the official hydrargyrum cum cretâ). It is adapted to both acquired and hereditary syphilis, and brings on effects of the most satisfactory nature. I am more inclined to use it in cases of the latter nature, but Mr. Jonathan Hutchinson is a great advocate of its employment in the former. In my experience, its use in the acquired form is not so satisfactory, as patients object to a powder when they think a pill may as easily be given. Whilst this is not a valid objection, it is one which in many instances should be heeded. In doses of from one to three grains thrice daily it acts well, but is apt to disturb the stomach and bowels in a very short time. It is well to watch its effects.

As in the case of liquid mercurial preparations, so with powders. We are suddenly brought to a stand-still by their paucity. The gray powder is the most useful member of its class, and you will find it the best in the hereditary form. Add a little sugar to your powder and children will ask for it, and the good results following its administration will be both marked and rapid.

We now come to the form in which mercurials are most commonly administered, and one in which the drug can be controlled as well as in any other. It is a peculiarly convenient method for the treatment of men who are in business or who have to travel. It obviates the necessity of carrying a bottle or spoon or measuring-glass. There is another thing to which I wish to call attention: As you know, pills in a box sometimes rattle and attract the attention of every one. Instruct your patients to crumple a piece of soft paper or take some absorbent cotton and place over the pills. This will deaden all sound and prevent impertinent interrogation on the part of friends or curious persons. Pills in a bottle will rattle; besides, the bottle is apt to be broken. If carried loose in the pocket the pills may melt, get broken, or distorted. This is merely a matter of detail, but one which will be appreciated by those who carry the pills. It should also be remembered that the majority of patients prefer pills because they are so made now as to be devoid of all taste, whereas liquid preparations are always more or less disagreeable if not positively nauseating. The solubility of pills is now beyond all question, owing to improved pharmaceutical methods. Thus the old prejudice has disappeared and pills have grown very much in favor with patients and physicians alike; more especially with the latter, on account of the greater accuracy of dosage which can be attained.

Of pills there is quite a large number which depend upon the salt of the metal which is put in that form. Those which enjoy the greatest popularity contain the proto-iodide. Some syphilologists use almost nothing but this, and yet, in my experience, it cannot absolutely be depended upon to give the best results. Dr. E. L. Keyes, of New York, finds what a patient's tonic dose of this salt is, and continues him on it until a cure is effected or complications arise which call for more energetic measures. He is a strong advocate of the "tonic" treatment, however, and asserts that he has obtained the best results from its employment. In my experience, I have found that, while it is beyond doubt the best mercurial to give to syphilitic women, in men it does not seem to act as well as other preparations, and for that reason I have abandoned it in their case. In women a quarter-grain pill thrice daily acts very nicely, whilst in men very little satisfaction will be derived unless a half-grain be administered. Even then symptoms will crop out which require

more energetic treatment, and resort must finally be had to a course which, if adopted from the beginning, would have been successful and not attended by any such unexpected or disagreeable complications. A much better pill, more especially for men, is that composed of biniodide of mercury. It acts in a more satisfactory manner and is much more active than the proto-iodide. This is easily borne in quarter-grain doses and rarely produces gastric irritation. It may bring on diarrhœa, which can easily be checked by simple astringents. I have mentioned these two pills on account of their popularity, although they can hardly be counted as mercurial, containing as they do a certain amount of iodine. Still, as it is really the mercury which does the work, it may be well to include them in this class.

A purely mercurial pill which was formerly very popular is that composed of blue mass, the pilula hydrargyri of the United States Pharmacopœia. It is, however, badly borne, as it must be given in at least grain doses, and readily irritates the intestines and rapidly brings on diarrhœa. It is rather unreliable in its action, the curative effects not keeping pace with the untoward ones. It is used very little now in view of the better preparations from which to choose. For the same reasons the calomel pill has been dropped by those who have had any experience in the treatment of syphilis. It is a treacherous remedy, and often must be discontinued at a critical moment, which disadvantage hardly recommends the mild chloride as an antiluetic. The bichloride of mercury pill is not so objectionable. Good results may be obtained from it, and but one drawback attends its employment,—namely, it is quite irritating to the stomach, and consequently must be used very carefully. An eighth of a grain is a good working dose, which may be increased or diminished according to the exigencies of the case. Of late years a preparation of mercury has been introduced which is made in pill form and enjoys a certain amount of popularity. This is the tannate of mercury, which possesses the peculiar property of being insoluble in acid but soluble in alkaline media. For this reason it is indicated in cases in which the stomach is irritable, the solution taking place in the upper part of the intestine. It is not as active a preparation as either one of the iodides, but it will give good results, especially if taken in one-grain doses three times a day. It may safely be administered before meals, as

it does not irritate the stomach in the least; nor, so far as my experience goes, does it irritate the intestinal tract. However, despite these good qualities, I would not advise you to place your reliance on this preparation alone. It is good, but not entirely reliable for what I call a final effect.

I have spoken to you of pills, but all the preparations I have mentioned are not necessarily in pill form. Pills may be made by druggists or by manufacturing pharmaceutists. I prefer the latter, because they are more accurately dosed, look better, and keep better. These latter are usually coated. Again, some manufacture the so-called tablets, which may be friable or compressed. The former are more soluble, but disagreeable to take. The coatings of pills may be sugar, gelatin, chocolate, or keratin. Concerning the relative advantages of each very little can definitely be said, as the matter is yet *sub judice*. It is only of the latter that anything can be formulated. The keratin-coated pill is insoluble in the stomach. Keratin, as you are aware, is insoluble in acids but very soluble in alkalies. This naturally indicates that it is useful in cases in which certain mercurials cannot be borne by the stomach, but which, nevertheless, should be exhibited. This has been a veritable boon to the profession, and has enabled physicians to attain results which were next to impossible before its introduction. Before closing I must not forget to call your attention to another form in which solids are administered,—namely, capsules. They are not among the most popular forms with patients, but exigencies sometimes arise compelling their use. The peculiar taste of some patients must occasionally be catered to in order to have them take remedies.

I have made no allusion to the correctives combined with mercurials by some physicians. These are opium and hyoscyamus. I have never found it necessary to use them, as the choice of available mercurials is so large that there is no real necessity for their employment. It is dangerous to use opium, because a habit may easily be acquired by the patient, the imagined benefit increasing the danger a thousandfold and perhaps ending in the destruction of an unfortunate.

III.

There yet remain several methods of mercurial treatment of syphilis, some of which are popular and others highly commended.

Do not be led astray by glowing accounts of certain methods which have been found inefficacious after a fair trial, but which are still lauded by those who have introduced them. Enthusiasm is to be commended, but cool judgment is a much safer guide for him who desires to achieve success. Do not depend too much upon hospital results, for, as I noted in a former lecture, hospital patients are susceptible of control such as no private one is, and they are not kept long enough to enable any one to draw final conclusions of any value. A private patient will call attention to the slightest shortcoming, and will insist upon better treatment if that which he is undergoing is not satisfactory. Whilst enthusiasm for any method of treatment is to be commended, it must not be pushed to an absurd conclusion. Careful observation, untrammelled by prejudice, is the surest as well as the safest guide. It is well not to abandon a measure because of one or two failures; but it is inexcusable obstinately to persevere in a course which is entirely irrational. What I wish to speak of to-day is the hypodermic, intramuscular, and intravenous injections of mercury in the treatment of syphilis. These have many enthusiastic supporters, but I would caution you against too much of this, and will give you my reasons as I go on.

You will find that one of the prerequisites for these injections is aseptic instruments. Except in special cases, it matters but little what form of syringe you use, provided it is surgically clean. The injected material must also be aseptic, and, unless these conditions are faithfully observed, abscesses and other disagreeable conditions are apt to supervene. The injections employed are soluble and insoluble. The most popular among the soluble injections is corrosive sublimate, made such by Lewin, who devoted much time and attention to it. He advocated doses of a centigramme, repeated twice a week, or, if preferred, smaller ones, administered more frequently. One of the chief objections to this salt is that its administration in this manner is quite painful. I desire, in this connection, to warn you against the almost universal custom of giving a hypodermic injection by pinching up the skin and then introducing the needle in the fold. The proper manner is to introduce the needle at right angles to the skin, and when it is traversed, incline it at a more acute angle and drive the piston home. This can all be done in one motion, and you will find that you make a much better injec-

tion than by the other method. You reach a lymphatic space instead of driving your solution into the connective tissue, and naturally obtain a better and more rapid result.

But to resume the consideration of the soluble mercurials which are utilized in hypodermic injections. I do not intend to speak of all the forms of mercury which have been employed for this purpose, for their name is legion. The salicylate of mercury, introduced by my friend, Dr. Silva Araujo, of Rio Janeiro, has been highly spoken of by a number of syphilologists. It is to be administered in the dose of two grains daily. In this dose it may be given for a number of months. Whilst an apparently valuable remedy, the salicylate of mercury has not become popular to any marked degree. It and many other preparations, such as certain composite preparations of mercury, have been highly lauded by those who introduced them and then dropped out of sight altogether. Thus, the mercuric albuminate was rapidly relegated to oblivion upon the death of its introducer. We may place in the same class the hydrochloride gelatin-peptone sublimate and the members of the so-called antiseptic group, comprising, among others, the carbolate, thymolate, and benzoate of mercury. The amide group has not met with much favor, despite Liebreich's recommendation, and it will be sufficient simply to mention them. The principal members of this group are the glycocoll of mercury, the succinimide of mercury, and urea-mercury. These salts are all accompanied by disadvantages which are sufficient to preclude their use in any but hospital practice. There is one soluble mercurial salt, however, which I can safely recommend as most excellent in the treatment of syphilis, and that is the bicyanide of mercury. Its administration, however, is very painful, unless some corrective be added which, whilst not interfering with its action, obviates the necessity of the patient suffering so much. I have found it to be a remedy which is easily borne and which gives most excellent results. The formula which I generally use is as follows:

R Hydrarg. bicyanid., 0.6 part;
Cocaini muriat., 0.04 part;
Aquæ destillat., 30.00 parts.—M.
Sig.—Use for hypodermic injection.

Of this I inject twenty minims daily until good effects are observed. Then the same amount is injected every other day, and the

frequency is diminished *pari passu.* You will find that, on the whole, this is the most reliable and satisfactory preparation of mercury to use in the treatment of syphilis. There is one precaution to observe in injecting this salt, and that is the proper choice of a good location. Some physicians have complained of dizziness and other phenomena appearing after its injection, but I have never observed them. This is probably due to the fact that my rule is to inject the solution in the back along the borders of the scapulæ, alternating daily from the right to the left side. Small details such as this must be carefully attended to if good results are to be expected.

The insoluble mercurials have enjoyed a large amount of popularity for hypodermic use in syphilis. The most highly vaunted has been calomel; but, before referring to it, mention should be made of injections of metallic mercury, which never gained any warm adherents on account of the bad effects following and the necessity of cutting out the injection nodules which were produced. More successful have been the calomel-vaseline injections, which, whilst made deep into the buttocks,—some two and one-half inches,—possess the advantage of not having to be made more than once in a week or ten days. Instead of liquid vaseline, olive oil, oil of sweet almonds, or glycerin may be used. In any event, the excipient must be aseptic. Whilst theoretically, because of the slight degree of trouble involved, this method is favored by the great majority of physicians, there are many who will not employ the remedy on account of the abscesses which so frequently follow its application, though I have never observed them in thousands of injections of the bicyanide. This liability to the formation of abscesses is one of the greatest objections to the use of the insoluble mercurial salts hypodermically. My friend, Dr. Xavier Watrazewsky, of Warsaw, introduced the injection of yellow oxide of mercury in the treatment of syphilis. He has gone so far as to state that three injections were sufficient for a cure,—meaning, no doubt, a course of treatment. Considering the fact that he uses such a small amount, this could not be predicated. The yellow oxide is rapidly absorbed, however, and lingers in the system for a long time, thus continuing to produce its effect. Black oxide of mercury and cinnabar have had their advocates for hypodermic injection, but have rarely been employed, as they are less energetic and efficient than some others I have mentioned.

I could continue to dilate upon more hypodermic methods, but I desire to say a word or two on intramuscular and intravenous injections. I have already hinted at the former in speaking of the use of the insoluble mercurial preparations. They are usually injected into some large muscle, such as the gluteus, and deposited there to form a deposit from which mercury is constantly absorbed and distributed throughout the system. That is the theory, but you will readily see its defect, which lies in the fact that the accurate dosage of the amount absorbed cannot be established. It is probably for this reason, as well as the occurrence of abscesses, that the injection of insoluble mercurials into the muscles has not become more popular. The intravenous injection of mercurials has necessarily been limited to the soluble preparations. Baccelli, who originated the method, was very enthusiastic over it, yet it has not been spoken of so highly by others. In fact, experiments on animals have resulted so disastrously as to preclude its adoption. The only argument which can be advanced in its favor is that the remedy is thrown directly into the blood-torrent, which, to my mind, is inferior to a mode which introduces it into the lymph-spaces.

So far as the injection method of using mercury in syphilis is concerned, it is one which has particular indications and is not adapted for routine treatment. There is no doubt that it is quick in action, producing results but little short of marvellous. On the other hand, these are not permanent. An apparently cured case is merely one placed in abeyance, and the course of injections must be followed by the internal administration of mercurials. Yet injections are sometimes of the greatest value in cases of emergency when nothing else will make an impression. They frequently produce good effects when the limit of tolerance has been reached by other means, and certain symptoms persist in spite of the best-directed internal treatment or inunctions. Whilst it is not the most judicious plan to resort immediately to injections in every case, yet the temptations for their adoption are numerous. The method is absolutely reliable, for the reason that the physician has complete control of the treatment. He knows how often it is administered, and is certain not only of the dose of mercurial which the patient takes, but also of the fact that he does take it. The method is one that can be absolutely managed and controlled, which cannot be said of any other in private practice. Another advantage

which I have noted in connection with hypodermic injections is the comparative ease with which parasyphilitic phenomena are relieved. The rapidity with which nervous phenomena yield to this treatment is nothing short of marvellous. I have observed cases of syphilitic insanity, with various delusions, hallucinations, and impulses, yield with comparative ease and in a relatively short space of time. Cases of various paralyses also act in a similar manner and respond very readily to injections. The same may be said of neurasthenias, hyperæsthesias, and analgesias. Another class—the so-called tertiary phenomena—also succumbs to the treatment without difficulty.

I have dwelt in detail upon the systemic treatment of syphilis with mercury because there is no doubt whatever that it is the true method. Hundreds of vegetable and other metallic substances have been introduced from time to time and highly lauded, from the sacred wood, guaiac, to the sulphate of copper, but as yet none has proved a succedaneum for mercury. From the most remote times of which we have any records up to the present day it has been regarded and has proved itself to be a specific for lues. The numerous modern vegetable alteratives so highly spoken of are from a therapeutic point of view naught but Dead Sea fruit. They cannot produce the desired results, and are useful only to enable patients to rest from mercury. You may have asked yourselves the reason of my speaking of the systemic use of mercury in the treatment of syphilis, and I will try to tell you why I was led to do so. I have observed that there is a great tendency, more especially in hospital practice, to treat all syphilitic patients with potassium iodide. By using a saturated solution of the salt it is an easy matter to order it in drops and thus save a great deal of trouble. But any one may observe that patients do not improve under this treatment, and to those acquainted with the therapy of the disease there is nothing strange in this. What I desire to impress upon your minds is the great truth of syphilology announced some years ago by a Frenchman: mercury alone will cure syphilis, potassium iodide alone cannot. Keep this always in mind and you will not go far astray in your treatment. I have treated many cases without ever having had recourse to the iodides, and my efforts have been rewarded by the best possible results. The iodides doubtless act upon the glandular system and in that manner possess a certain amount

of power in eliminating; they also conduce to reduce the amount of new connective tissue which forms; but they cannot accomplish the principal thing which mercury never fails to do. They cannot destroy those small round cells so characteristic of the syphilitic process. And after the iodides have accomplished all of which they are capable, you will still be compelled to give mercurials to round out completely the treatment and make it perfectly satisfactory.

For this reason you will find it much more satisfactory to adhere to mercury and not run after every new preparation which may be lauded in specious terms. It behooves every one to be wary of those wonderful preparations which, upon a fair and full trial, prove to be nothing but snares and delusions. Any one who has practised a quarter of a century has seen the temporary rise and permanent fall of numbers of these highly vaunted specifics (?). It is in this matter more especially that the old way is the best. I do not mean by this the preparations and doses used a half-century ago, but rather the principles of treatment and the therapeutic agents which are employed. We are too progressive at the end of this century to stop and beat time. But progressiveness does not necessarily imply iconoclasm. Improvement is possible without destruction, and it is our function to be always progressive and to hold fast to that which is good.

TREATMENT OF THE COMPLICATIONS OF THE SYPHILITIC CHANCRE.

A CLINICAL LECTURE DELIVERED AT THE EAST END DISPENSARY.

BY A. FOURNIER, M.D.,

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Gentlemen,—The three principal complications of the syphilitic chancre—inflammation, gangrene, and phagedæna—are closely connected with one another, and in fact frequently depend on the same cause. Thus, excessive inflammation often terminates in gangrene, and gangrene, in turn, may be a commencement or a form of phagedæna.

For this reason the remarks that I am about to make concerning the therapeusis of these complications are applicable to them all as a group, and I shall begin with the most general one, so as not to have to repeat it in connection with each of the subheadings that I intend to make on the subject.

Whenever you have to do with a syphilitic chancre complicated by inflammation, gangrene, or phagedæna, you should, before treating the chancre itself, look for the cause to which the complication is due and, if possible, remove it. It is quite customary to see the complication disappear in a short time, and the chancre return to its normal aspect and conditions when this has been accomplished, without any treatment of the complication in itself. Let me give you an instance.

You have only to recall the condition in which our hospital patients come for consultation, their chancres inflamed, purple, swollen, ulcerated; in a word, on the high road either to gangrene or phagedæna. Then see those same chancres after the patients have been under treatment three or four days; almost invariably you will find them completely transformed into chancres of the usual type. Now, what is the secret of this metamorphosis? Sim-

ply that the chancres have been cleaned, dressed, and protected from the causes that had aggravated their condition,—namely, neglect, dirt, friction, etc., as local irritants, and fatigue, bad food, etc., as general causes.

Cases such as the following are sometimes met with. A patient with a syphilitic chancre will treat it altogether too vigorously. He follows the advice of every friend. One recommends silver nitrate, which he applies; another suggests perchloride of iron, which he also uses; until he finally comes to your office with his chancre inflamed, painful, spreading, and in imminent danger of gangrene. You need then only point out the cause of the trouble; tell him to stop all cauterizations, to bathe frequently, to keep the chancre well washed, and to apply some harmless salve; as a result the chancre will in a very short time resume its normal aspect.

Not long ago a patient came to me with a hard chancre complicated by gangrenous balanoposthitis, the penis looking like a club, or tongue of a bell; the prepuce was perforated and the glans partly protruded, while on it could also be seen a patch of gangrene. The prepuce was immediately sectioned and laid open, and in the furrow back of the glans was found a lump of lint that had been forgotten by the patient! Removal of this foreign body put a stop to the gangrene and phagedæna, and recovery was rapid.

In the same manner whenever gangrene and phagedæna depend on paraphimosis they can be cured at once by an incision that removes the strangulating pressure.

This point is therefore self-evident. Whenever a cause for the complication is found, your first duty is to remove it. There will of course be cases in which this may escape detection, and there will be others in which the cause, even if found, cannot be removed, as when phagedæna depends on old age, glycosuria, pregnancy, or cachexia: but in the great majority of cases properly directed treatment will be successful.

With the close of these remarks concerning generalities I come to each complication in particular.

1. *Inflammation.*—This is the commonest as well as the chief complication of the hard chancre, and the one that must be treated first, as, if unchecked, it may lead to balanoposthitis, vulvitis, gangrene, or phagedæna. The means at our disposal for this purpose

are: (*a*) Rest, relative or absolute, according to the degree of the process; in serious cases confine the patient to bed. (*b*) Bathing. There is nothing more efficacious than this when it is energetically employed. General baths, with ordinary water or bran water, every day; in serious cases baths lasting an hour or more, at a temperature of 35° or 36° C. As a help to this, local baths of the penis in any appropriate vessel and continued for from five to twenty minutes. (*c*) Moist dressings applied to the penis; for women, similar applications to the vulva. (*d*) Methodical dressings with the most harmless and least irritating preparation possible; for instance, absorbent cotton dipped in boiled water or in a decoction of poppy-heads.

2. *Phimosis.*—When a hard chancre has caused inflammatory phimosis, it must be treated in the way I have suggested,—namely, rest, baths, and fomentations. Two complications may present themselves, if the chancre is hidden beneath the swollen prepuce.

When phimosis is absolute the case is simplified, as it is impossible to dress the chancre itself; all that can be done is to reach it by means of injections beneath the prepuce. These are very necessary, and have for their object the prevention of balanoposthitis, which may result from the stagnation of the secretions of the chancre beneath the foreskin. I will refer again to these injections in connection with the treatment of balanoposthitis. Many solutions have been used for this purpose; the best is a weak solution of silver nitrate (1 to 100 or 1 to 200), used two or three times a day.

When, on the other hand, phimosis is incomplete, and the patient can uncover the glans, though with difficulty, the question is not so simple. Are we, under these conditions, to treat the chancre as in the preceding instance,—that is to say, as if phimosis were absolute; or are we to pay no attention to the phimosis, and treat the chancre as we would an ordinary one situated beneath the prepuce and uncomplicated by phimosis? Everything depends on the state of the case and the degree of inflammation of the prepuce. If the patient can draw the foreskin back with such ease as to render it certain that there will be no difficulty in bringing it forward again, the chancre should be dressed in the ordinary way, though the patient should be warned of the possibility of paraphimosis, and that the treatment must be abandoned should there be the slightest difficulty in its application. When, on the other

hand, there appears to be trouble about getting the foreskin back over the glans again, treat the case like one of absolute phimosis. There is no real objection to treating a chancre beneath a foreskin as though phimosis existed; whereas it is highly undesirable to complicate a chancre with paraphimosis.

3. *Balanoposthitis.*—This is a complication that must be combated by every means at your command, as it is often the cause of the most serious accidents connected with the chancre,—that is to say, gangrene and phagedæna. From the point of view of treatment it has two main forms: the average case, and the serious, acute form with gangrene.

The treatment of the average form consists in the antiphlogistic treatment of chancre complications as already stated, and particularly in local and general bathing and fomentations, together with irrigation beneath the foreskin. The importance of the latter means may be seen at once. This irrigation must be carried to the full depth of the preputial sac by means of a soft red catheter (No. 14 or 15, French), which should be moved around until the entire cavity has been cleansed.

Two kinds of solutions should be employed, the first simply for flushing purposes,—boiled water or boric solution; this irrigation should be continued until all the pus and filth has come away and the solution returns absolutely limpid. When this has been accomplished a modifying solution should be used, and experience has shown that silver nitrate is the best,—a solution varying between one-half and two per cent. Inject a couple of ounces two or three times a day, and when suppuration is profuse, repeat the flushing irrigation two or three times a day also, to avoid stagnation beneath the foreskin.

In the serious form of balanoposthitis with threatening gangrene, which is announced by the fetid odor of the discharge, or with gangrene already present, the danger is a real one, and prompt and energetic action is imperative. The only thing to do in these cases is to open up the cavity of the prepuce entirely, which may easily be done by making with a pair of scissors a vertical incision of the whole prepuce. The two flaps laid open, all the subjacent surfaces are exposed to view. Clean them carefully, dress the chancre with iodoform, and cover the whole with iodoform gauze. On the following day begin the bathing process. The usual result

of these measures is to stop entirely the inflammatory complications and gangrene.

4. *Paraphimosis.*—When this complication occurs one of two things must be done. If there appears to be a chance to get the foreskin over again,—and this is possible more frequently than one would think, if you proceed methodically, after making a number of punctures with a sterilized needle and kneading and pressing the tissues,—you must try to, and you will pretty generally succeed in the early stages.

If, on the other hand, the chancre appears to be suffering from the disorder to the circulation created by the strangulation; if it is swollen, œdematous, and of a purple color, or if it shows a tendency to gangrene and phagedæna, the indication cannot be avoided, and you must get rid of the strangulation, which can readily be accomplished by an incision on the upper part of the penis.

5. *Gangrene and Phagedæna.*—These two complications can be treated together, as their therapeusis is about the same.

Absolute rest in bed is, of course, indispensable, and, in addition, the following directions must be observed:

(*a*) Use every effort to ascertain the cause of this complication. I have referred to this point before. Many cases of phagedæna depend on very manifest causes (inflammation, balanoposthitis, phimosis, and paraphimosis), and the simple removal of these insures recovery.

(*b*) It would puzzle me to tell you just how the process of bathing acts on gangrene, and particularly on phagedæna, whether it lessens nervous irritation by reducing inflammation, or whether it modifies the diseased tissues in some other way. In any case, it is one of the most active means we possess against these two serious complications. In many cases it has given me such excellent results that I have been led to assign to it a most important rank among our therapeutic means. But to succeed it must be applied in a certain way. Frequent and prolonged baths must be given, three hours at a time, at a temperature of 35° or 36° C., every day, though at the end of a week, if distinct improvement is noted, a bath of two hours every second day will be sufficient. With these should be combined local baths of about twenty minutes' duration before the time for dressing the chancre.

(*c*) As a local dressing for the chancre nothing equals iodoform

in either gangrene or phagedæna; every one agrees as to its modifying influence in these circumstances. In cases of gangrene or gangrenous phagedæna, when the tissues are not very irritable, it is best to apply the drug pure, cover with cotton, and fix in place with a bandage; but when dealing with red, inflammatory phagedæna, which is much less tolerant, the iodoform should be mixed with lard, vaseline, or lanolin, and begun with small doses (for instance, 1 in 10). In more than one case I have seen this form of phagedæna revolt against dry dressings and iodoform in powder, and tolerate nothing but ointments. Pure iodoform can be given often only at a later stage.

In this connection account must be taken of different idiosyncrasies. Phagedæna is a capricious disorder, and tolerates or refuses to tolerate a given remedy according to the patient and the conditions. With some patients iodoform burns for a while, or irritates, and must be set aside for the time being,—not to speak of the idiosyncrasies where it causes deep local eruptions that necessitate its abandonment altogether. This holds equally true with almost all remedies; when a phagedæna rebels against one drug, it is apt to do so for almost any remedy. I have seen instances in which *everything* made the trouble worse, and where I have been obliged to abandon all forms of active treatment and confine myself to boiled water or lard. As a rule, however, this universal intolerance lasts only for a given period, and there comes a time when the usual treatment can be again applied.

Finally, as regards gangrenous phagedæna, I may say that when the inflammatory symptoms are passed benefit will be derived from applications of tincture of iodine twice a day.

(*d*) Whereas the general treatment has usually but slight effect on the ordinary form of chancre, it has an energetic action on phagedæna. Experience has taught us this, and it has also taught us that the mixed treatment will succeed best. You must, therefore, give both mercury and iodide, and in full doses: 0.1 gramme (one and one-half grains) of protiodide, for instance, and four, five, or six grammes (sixty to ninety grains) of potassium iodide *per diem*.

In one case of red phagedæna of a lingual chancre (a very extensive chancre with a most deplorable appearance) I got a remarkable result from injections of 0.05 gramme of calomel; I

shall never forget the change produced by the first injection, and shall certainly use this remedy again under similar circumstances.

Such are the means we have at our disposal against chancrous phagedæna, and I think that the therapeusis of this serious complication is comprised in the following terms: balneation, iodoform, and the mixed specific treatment.

Having said this much about what *is* to be done for phagedæna, it may not be amiss to add a few words about what is *not* to be done. Syphilitic phagedæna must not be treated by means of cauterization. All chemical caustics, as well as the actual cautery, have been used, but with deplorable results. To destroy entirely this process would necessitate the destruction of a large amount of tissue,—greater, usually, than would have been caused by the phagedæna itself. In a word, it would be necessary to destroy a large portion of the penis or vulva, and this is not to be thought of. It has been asserted that without actual destruction the red iron may modify the process; but I have never seen such modification do anything else than intensify the disorder, and I think the idea should be totally abandoned.

Counterirritants must not be used. Such applications only make the phagedæna worse. This is likewise true of cauterizing by silver nitrate, which, unfortunately, is so frequently done to the hard chancre and to its complication, phagedæna. I recall a number of cases of phagedæna due, without doubt, to the many times an ordinary chancre was touched with nitrate of silver.

I wish also to warn you against the use of the multitude of new preparations with which we have been flooded of late years. Some of these may eventually become very useful, and put our old acquaintance iodoform in the background; but until further notice it will be well not to make too many ventures, and in treating such a serious disorder as phagedæna you had better confine yourselves to iodoform, which has always answered so well.

My final recommendation is not to be in a hurry to remove pieces of dead or mortified tissue in gangrenous chancre or phagedæna. The temptation to clean up a region is very great, but is a mistake in this case. In the midst of an apparently dead piece there may still be permeable vessels which will cause a hemorrhage, not to speak of the risk from absorption by opening vascular tissue surrounded by a gangrenous region.

Medicine

RECENT ADVANCES IN DIAGNOSIS.

A RÉSUMÉ OF PRACTICAL POINTS BROUGHT OUT IN THE DISCUSSIONS IN THE MEDICAL SECTION OF THE THIRTEENTH INTERNATIONAL MEDICAL CONGRESS, PARIS, 1900.

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CYTODIAGNOSIS.

Professor Widal, of Paris, presented the results of a series of microscopic investigations of the cell-contents of various effusions into the serous cavities of the body. He has found that characteristic pictures exist for different pathological conditions, and, as the result of his investigations, he has been able to formulate a set of rules that will guide the clinical observer in the diagnosis of certain conditions of serous cavities which have long been a source of rather anxious uncertainty.

The examination of the serous fluid from the pleural cavity is made after defibrination. The fibrin is removed either before or after coagulation. A drop of the fluid is taken, preferably on a platinum needle, and is spread over an object-glass by movements in a circular direction. Different specimens are then colored with thionine, eosine, hematein, and with Ehrlich's triacid solution. The character of the cells that are found to predominate gives important information as to the etiology of the effusion.

In the form of pleuritis which has long been known as idiopathic pleurisy, or pleurisy from cold, Landouzy showed, by a series of microscopic investigations and inoculation experiments, that the effusions were really due to tuberculosis. In this form of pleurisy a number of small lymphocytes are found exhibiting a marked tendency to run together. Besides these a number of red

blood-cells are often found. Other cells are very rare. On the other hand, in pleurisies that occur in frankly tuberculous subjects—that is, patients who have easily recognizable tuberculous lesions of the lungs, with an extension to the pleura and consequent effusion—the liquid withdrawn from the pleural cavity presents a very different cell-formula. As a rule, in this fluid but very few cells are present. A few red cells occasionally occur and a few small lymphocytes may be found. The principal form of cell that occurs, and even this is rare, consists of old deformed polynuclear leucocytes. In these the nucleus is found much disturbed in form, and the protoplasm of the cell contains a number of degenerate-looking neutrophilic granulations. At times a very few large mononucleated leucocytes more or less deformed are found. Agglomerations of cell-elements amorphous in character and without nuclei may also be seen.

Mechanical pleurisies, such as occur in cardiac cases or in patients suffering from Bright's disease or cancer, or that develop as the result of compression or irritation in neighboring parts, are characterized by the presence of large endothelial cells, evidently the result of desquamation from the surface of the serous membrane. Their enormous dimensions, as compared with the red cells and with the lymphocytes which sometimes surround them in considerable numbers, make them recognizable as soon as seen. They are either single or grouped together by twos or threes, sometimes even by fours, and so form the pathognomonic characteristics of this type of pleurisy. At times sheets of these endothelial cells occur, representing a veritable endothelial desquamation. These cells may retain their connection with one another for a long time after the effusion has taken place.

In the effusion from serofibrinous pleurisy due to the streptococcus a number of polynuclear neutrophiles with deformed nuclei are found, but the picture is scarcely characteristic. Pneumococcic pleurisy presents a histological formula which can scarcely fail to produce the impression that the tissues are vigorously reacting against an infectious invasion. The effusion is characterized by the presence of red cells and of some lymphocytes, but especially by an abundance of polynuclear leucocytes. The pathognomonic sign, however, is the existence of a number of large mononucleated cells, mostly of endothelial origin, some of which are

veritable giant phagocytes. These will be found to englobe even the polynuclear cells in their protoplasm. It is very rare that one finds two or three of these endothelial cells still retaining their connection with one another.

Pleural effusions which occur during typhoid fever are frequently hemorrhagic. The presence of blood in the effusion makes it difficult to say what is the exact cellular formula of the liquid. In most cases there seems to be a predominance of large mononuclear leucocytes. Twice during typhoid fever it has been noted that a majority of the cells in the pleural effusion were eosinophiles. In a third case in which a large number of eosinophiles were found the pleuritic effusion occurred during the course of a tuberculosis. In these cases the eosinophiles in the blood were counted, and it was found that there was a larger proportion of these cells in the pleuritic effusion than in the blood of the patient.

The liquid taken from a pleurisy can be as easily sent to a laboratory for examination as can the sputum of a suspected consumptive. The information that the histological examination of the fluid may furnish is almost as valuable as that given by the bacteriological examination of the sputum. There seems no reason to doubt, then, that this new method of diagnosis will prove of great service in certain extremely difficult cases. Besides its application to the serous effusion of pleurisy, cytodiagnosis gives valuable information as to the cause and character of effusions into other serous cavities.

The serous fluid withdrawn from a hydrocele contains in ordinary cases only simple endothelial cells. This is a sign that the pathological condition is mainly mechanical in origin. At times a certain number of red blood-cells will be found in hydrocele fluid. In these cases careful inquiry into the history will usually elicit the fact that trauma of some kind has recently led to an increase in the size of the hydrocele. Very rarely other cells, especially polynuclear cells, may be found, and then they constitute an evidence of the presence of some infective process, though this may be but slight and have given but very few inflammatory symptoms.

From a number of joints of patients affected by acute articular rheumatism serous fluid was withdrawn for purposes of examination. In frank rheumatic cases the predominant cells were always

polynuclear neutrophiles. These are the cells which, according to our present knowledge of infection and reaction, represent a phase of nature's protective processes against the invasion of infectious material. The fluid withdrawn from these rheumatic joints was carefully inoculated on all the media known to modern bacteriology. In not a single case where proper precautions were taken against contamination did any growth appear on these media. While the cellular formula of the arthritic effusion in acute rheumatism, then, makes it clear that rheumatism is an infectious disease, it is evident that its microbic cause is of a kind different from any microbe with which we are at present familiar. It may be, of course, that the micro-organismal cause of rheumatism, while evidently parasitic in nature, is not saprophytic,—that is, will not grow on any but living organic matter.

Cytodiagnosis enables the clinician at once to distinguish between rheumatic and traumatic effusions in the joints. In cases of trauma the joint-fluids always contain some red blood-cells, some endothelial cells from the lining of the serous cavities of the joints, and some few lymphocytes, but do not contain any polynuclear cells unless some infective process has been superadded to the trauma. Tabetic joints furnish a fluid which very much resembles that found in cases of traumatic arthritis. This serves to substantiate the theory that tabetic arthritis is really due to trauma or a series of traumata consequent upon the incoördination of the patient and the fact that the disturbance of sensation causes neglect of the usual precautions against needless contact of the surface of the joint.

The cytodiagnosis of the cerebrospinal fluid has, of course, been the subject of investigation by a number of observers in recent years. In cases of tuberculous meningitis Widal found that the small lymphocytes predominated in the fluid withdrawn by lumbar puncture. At times, when it is impossible to find bacilli in the fluid owing to their rarity, the predominance of small lymphocytes exhibiting a marked tendency to run together is almost pathognomonic of a tuberculous process in the central nervous system.

SERUM DIAGNOSIS OF TUBERCULOSIS.

The necessity for some unfailing method of diagnosing tuberculosis at its very beginning becomes more insistent as we realize

more fully that it is at this period that the disease can be easily and completely cured. The discussions at the Congress showed that the use of tuberculin does not meet with as much opposition among French medical men as it does in Germany. A number of prominent clinicians continue to decide the knotty problem of incipient tuberculosis by the tuberculin test. Professor Arloing, of Lyons, insists that the serum diagnosis of tuberculosis can be made quite as easily and with as much assurance as can the Widal test for typhoid fever. He has tried his method of serum diagnosis on one hundred and ninety-one tuberculous patients, on one hundred and thirty patients suffering from other diseases, and on thirty-four individuals in apparent good health. He considers that these investigations have placed the question of the diagnostic value of the serum diagnosis of tuberculosis beyond all reasonable doubt.

He formulates the following rules for it. In the initial stage of pulmonary tuberculosis the agglutinative power of the blood-serum is constant, though it exists in various degrees. Sometimes the agglutinating power is manifest only when the dilution of the blood-serum does not exceed one to five; at other times agglutination is produced by a dilution as high as one to twenty. In serious acute cases with extensive lesions the serum reaction frequently fails or is very feeble. The agglutinative power of the serum of tuberculous patients seems to be in an inverse ratio to the gravity of the infection and the extent of the pathological condition.

In patients suffering from other than tuberculous affections, and in whom the ordinary physical signs and the clinical study of the cases reveal no sign of tuberculosis, the serum reaction frequently shows that latent tuberculosis is present. In many of these cases, in Professor Arloing's experience, the subsequent course of the disease or the autopsy has shown the correctness of the serum diagnosis. In a certain number of individuals who were apparently healthy, the agglutinative reaction for tuberculosis has proved to be present. This has not occurred, however, oftener than might be expected from what we know of the frequency of latent tuberculosis.

Dr. Arloing has tried the serum-diagnosis test in eighty cases in animals. The conclusions derived from it were found by autopsy to be exact in all except a single case. It is a question whether

this case may not have been one of those forms of pseudo-tuberculosis in which all tests fail,—in which even the tuberculin test, though this is accepted as infallible by all veterinarians, cannot be depended on.

A negative serum reaction, then, in a case of suspected tuberculosis is not final, because the agglutinative power is in certain cases not present. Where there are other positive signs of tuberculosis, the absence of the agglutinative power is of unfavorable prognosis. Where there are, however, no clinical signs of the presence of tuberculosis, a positive serum diagnostic reaction puts the case almost beyond question in the ranks of the tuberculous.

The cultures employed for serum diagnosis are those grown in a homogeneous glycerin bouillon. The test is a very simple one, can be done readily and quickly, is absolutely harmless to the patient, and certainly affords important information for the diagnosis of tuberculosis at a time when assurance of the presence of the disease means all the difference between cure and advance of the pathological process.

OTHER HINTS AS TO EARLY DIAGNOSIS OF TUBERCULOSIS.

It was pointed out that a knowledge of the family tendencies of a patient suspected of tuberculosis may furnish valuable hints for the early diagnosis of the disease. Papillon, of Paris, gave what he called the law of hereditary reaction. Its application is based on the fact that the toxines of tuberculosis present in the blood of a tuberculous patient, from the very beginning of the disease, cause symptoms in such organs as are especially non-resistant to irritation. In families, for instance, in whom a dyspeptic tendency exists, one of the first symptoms of beginning tuberculosis will be digestive disturbance. In families in whom cardiac troubles occur, palpitations, tachycardia, or even the presence of a temporary functional murmur will be noted. In families with neurotic tendencies the neurasthenia which is so often among the preliminary symptoms of tuberculosis will be especially noticed. If the family practitioner knows his patients well, there is no reason why these various symptoms, though seemingly quite distinct from the tuberculosis, should mislead him as to the fundamental pathological process. This law of hereditary reaction to the tuberculous toxines has a diagnostic value that is at least equal to many

other methods, and promises to be of distinct service in the early diagnosis of the disease.

Tuberculous Associations.—A factor in the production of tuberculosis that should be sought for quite as sedulously as an hereditary tendency to the disease is the patient's association with other people. It is now well known that tuberculosis is a house disease, and one of the questions that should always be asked a patient is not only whether any member of his immediate family has or has had tuberculosis, but whether any one living in the same house is suffering or has suffered from the disease. Where individuals work with many others, they should be asked whether any of their fellow-workmen have a chronic cough. A number of cases were reported at Paris in which tuberculosis occurred in a series of workmen or clerks employed side by side in the same factory or store.

RECENT URINOSCOPY.

There is a general agreement among clinicians that the present methods of examining urine are crude and defective. Much more attention is devoted to finding abnormal constituents than to the determination of the effectiveness of urine as an excretory product. What the clinician needs particularly to know is how much of its duty the kidney is performing and how much it is neglecting. Three recent methods that it is hoped will throw light on this important question were the subject of interesting discussion at the Congress.

Cryoscopy.—This is a method of determining the constitution of urine by finding its freezing-point. The name is derived from two Greek words, *κρύος*, ice, and *σκοπεῖν*, to examine. Raoult's law is well known. It may be stated as follows: the lowering of the freezing-point of a solution is proportional to the number of molecules dissolved in the unit of volume of the liquid, whatever be the nature and weight of the molecules. Of two urines, then, the one whose freezing-point is lower will contain more molecules in solution than the other. The value of urine as an excrementitious product depends on the number of molecules of dissolved material that it carries out of the organism. The number of molecules dissolved in the urine represents also, within certain limits, the permeability of the kidney. This principle is not absolutely true, because the sodium chloride dissolved in the urine cannot be

considered an excrementitious product: it passes out of the organism unchanged. Allowance has always to be made, then, for the number of chloride molecules present, and this can be done by estimating as accurately as possible the amount of the chloride in the urine.

Koranyi, of Buda Pesth, who first introduced the idea of cryoscopy to the medical world some three years ago, suggested that the ratio between the amount of chlorides in the urine and the amount of other solid substances dissolved represents the efficiency of the urine as an excretory medium. To substantiate this claim he recurred to the generally accepted theory of the physiological process of urinary secretion. From the glomeruli there escapes into the capsule of Bowman, by the force of osmosis alone, a watery solution of sodium chloride. As this solution escapes it is extremely dilute, having the same osmotic tension as the blood itself. Afterwards some of the water of this solution is reabsorbed, making the chloride of sodium solution more concentrated. As this concentrated solution passes through the urinary tubules it is joined by certain excrementitious substances from the blood. Here Koranyi introduces an idea that enables him to support his theory as to the value of cryoscopy in urinary examination. He says that a molecular interchange takes place between the chloride of sodium solution that filters out at the glomeruli and the blood when this solution reaches the canaliculi. The ratio between the amount of chloride of sodium in the resulting urine and the other substances in the solution represents the extent to which this molecular interchange has been carried. Where there is an excess of chlorides as compared with their usual ratio to the other molecules in solution, the urine does not represent an efficient excretory product.

The determination of the freezing-point of urine is not a difficult matter nor is the apparatus for the purpose expensive. A delicate thermometer is required, the scale of which can easily be read to at least tenths of a degree. This is placed in a test-tube containing the urine to be examined and is held in position by means of a perforated cork that fits into the tube. This test-tube is placed within another slightly larger and also held in position by means of a perforated cork. The two test-tubes are then set firmly in a stand-glass in which they can be surrounded by some volatile liquid, such as ether or bisulphide of carbon. The two

tubes may be held in position in the stand-glass also by means of a perforated cork. Through this cork there must be two other openings for the admission and exit of air. A current of air is then blown or drawn through the volatile liquid, with a consequent reduction of temperature, which can be read from the submerged thermometer when the freezing-point of the urine has been reached. From the fact that the tube containing the urine is not immediately in contact with the volatile liquid, but is surrounded by a layer of non-conducting air contained in the second tube, its temperature is reduced gradually; this is necessary in order to avoid error. At least ten minutes should be consumed for the operation, though recently in France five minutes have been considered sufficient.

The apparatus is not very cumbersome, and might easily be brought to the bedside, even in private practice. A flow of air through the volatile liquid may be secured by means of a water-tube exhaustion apparatus, which works on the principle that a column of water flowing freely in a tube carries with it air and so serves to draw a current of air through any space with which it has an indirect connection. To facilitate finding the freezing-point of the specimen being tested, Koranyi has suggested that when the temperature has nearly reached the normal freezing-point of urine (which is from about 1.25° to 2.2° C.[1] below that of water), a small piece of ice may be thrown into the urine through a branch tube provided for the purpose, and that the presence of this ice will cause the crystallization of the urine the moment its freezing-point is reached.

Practical Applications.—By means of cryoscopy Professor Koranyi has shown that in the urine of cardiac cases certain changes take place which afford timely warning of the approach of circulatory failure. These changes are of sufficient significance and the information they give is sufficiently direct to enable the physician to decide that his patient must give up his occupation, or even must take to his bed, although there may be at the moment but few symptoms of circulatory disturbance.

In renal affections the lowering of the freezing-point of the urine is always a favorable omen. The more nearly the freezing-

[1] The freezing-point of the urine differs for different individuals, but is practically constant in the same individual.

point approaches that of water, the less excrementitious material does the urine contain, and the more danger there is that the patient will suffer from the retention of toxic material in the blood.

In anæmia the freezing-point of the urine is never as low as it is in health. The relation between the urine of anæmic patients and that of normally healthy individuals seems to be about the same as that which exists between the specific gravity of the blood under the same conditions. The character of the urine depends not on the kidneys alone, but also on the condition of the blood, and the new method of urinary examination promises to show the relation between these two fluids as it has never been shown before.

Systemic Metabolism.—Professor Bouchard, of Paris, has used cryoscopy for another purpose than merely the determination of the number of molecules in the urine. He considers that it is a measure of metabolism.

The albumen molecule is the main constituent of animal tissues. The molecule of urea is the principal constituent of the solid matters of the urine and represents the completion of metabolic processes within the body. The molecular weight of the albumen molecule is 6000; that of the urea molecule is 60,—that is to say, one hundred times less. During metabolism the complex albumen molecule is gradually broken up into more and more simple molecules until it finally becomes urea. The more perfect metabolism is, the more nearly will all the excretory products be in the form of urea and the greater will be the number of molecules contained in the solid constituents of the urine. The freezing-point of the urine depends exactly on the number of molecules in solution. This is, therefore, an absolute index of the perfection of metabolic processes within the body. In determining this, however, the chloride of sodium molecules present must not be allowed to disturb the calculation. With them metabolism has nothing to do, since they enter and pass out of the organism unchanged. As in the former case, however, it is not difficult to eliminate this disturbing factor if we know the freezing-point of a solution of sodium chloride of a given strength.

Renal Permeability.—Two methods of testing the permeability of the kidney have recently been tried and are recommended by a number of clinicians. In many pathological conditions it is important to know whether the kidney is allowing excrementitious

materials to pass, or is inhibiting their passage and so causing the retention of toxic substances within the body. The iodides have been tried, with the idea of deciding, from the length of time required for an iodide reaction to be found in the urine, whether the kidney is allowing substances in the blood to pass freely or is delaying their exit from the body. The iodides, however, are too diffusible, and find their way through the kidney whether it is diseased or not.

Methylene-blue has been recently tried, and has been found to be a good index of renal permeability.[1] If methylene-blue is given in the ordinary dose of five grains, its excretion begins in from one-fourth to one-half hour. In pathological conditions its appearance on the urine may be delayed for an hour or more. Its excretion lasts from thirty-five to sixty hours under more or less normal conditions. All of the drug may find its way out in a much shorter time than this, but then there always exists an epithelial nephritis that makes the kidney too permeable. In rare cases the abridgment of the time of excretion may mean an excessive impermeability of the kidney, when very little of the drug is allowed to pass and urinary elimination ceases early, the blue finding its way out of the organism by other avenues.

When the time required for the excretion of the methylene-blue is prolonged, renal impermeability exists. This impermeability is greater the longer the time taken for excretion. The quantity of blue eliminated should go on increasing gradually until it reaches a maximum, and then should gradually diminish until it disappears from the urine. The occurrence of this cycle of elimination can be determined by examining the urine passed at suitable intervals. It gets bluer and bluer until between the thirtieth and fortieth hour, and then gradually has less and less color. If elimination does not follow this regular course, some diseased condition exists Irregularity of excretion often means that one kidney is diseased while the other remains healthy.

When renal impermeability can be demonstrated by delay in the elimination of the methylene-blue, there is always danger of

[1] Lepine recommends rosaniline trisulphonate of sodium for the same purpose; acid fuchsin has also been used. But neither of these substances possesses any advantage over methylene blue, and the experience with them is much more limited.

the development of uræmic conditions. It is well known how important it is to detect the preliminary symptoms of such conditions if successful treatment is to be hoped for. This new diagnostic method promises, then, to be of the greatest service in the prophylaxis of coma in cases of Bright's disease and of eclampsia during pregnancy.

Phloridzin is another substance which is used to determine renal permeability. When this drug is administered, sugar always appears in the urine. The rapidity with which glycosuria occurs and its amount constitute an index of the renal permeability. Nitze and Richter, in Berlin, have found this method of great service in renal surgery. When the removal of one kidney is being considered, it is important to know whether the other kidney is diseased. Phloridzin is administered and then the urine from each kidney is collected by means of ureteral catheterization. In the urine from the healthy kidney sugar will be found in abundance shortly after the administration of the drug; in that from a diseased kidney sugar does not occur.

This subject is of special interest from another stand-point, since it once more calls attention to the fact that the kidney has an influence in the production of glycosuria. It has been customary to regard the kidney only as an excretory organ for excessive sugar in the blood. There are evidently times, however, in which the disappearance of sugar from the urine and the consequent presumed improvement of diabetic cases are really not due to any amelioration of the metabolic faculty for sugar, but rather to some pathological condition of the kidney which prevents the excretion of the sugar, though it is present in the circulation as abundantly as ever.

CASES PRESENTING RENAL, CARDIAC, AND PULMONARY LESIONS.

ABSTRACTS FROM CLINICAL LECTURES DELIVERED AT THE PENNSYLVANIA HOSPITAL.

BY ARTHUR V. MEIGS, M.D.,

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Case I.—The specimens,[1] which I shall show you, were removed from the body of a woman who died after being in the hospital only about twenty-four hours, her admission and death having occurred between two of my visits. She had been under the care of a prominent physician, and was also in the hospital last summer from July to September. The records at that time show a diagnosis of acute myocarditis and acute dilatation, and that she left the hospital having recovered from her acute symptoms. She can hardly be said to have had fever, the temperature rising as high as 99° F. or even 100° F. at times, but falling again to 97° F. or, as on one occasion, as low as 96° F., the temperature being subnormal most of the time. It might, therefore, be called a temperature with little more than the normal variation.

The history indicated that the woman had organic disease of the heart. An examination of the urine showed it to contain albumen and numerous casts. During her first stay here there were also marked congestion of the lungs and hydrothorax, fifty-seven ounces of sanguinolent fluid having been withdrawn from the left pleural sac.

In regard to the heart, the apex-beat was found in the sixth interspace and two and one-half inches from the sternal line, the impulse being also felt below the xiphoid cartilage. The action of the heart was irregular; and a systolic murmur was heard at the apex, also at the aortic cartilage and over the vessels of the neck. I talked with the patient's physician, who told me that he thought

[1] Clinic of December 6, 1899.

there was also a presystolic murmur, which is not mentioned in the notes, and that he suspected mitral stenosis.

The heart before us shows a lesion which is very interesting, and one that is not often seen in Philadelphia, or in this country, in fact. The condition is that of the extreme form of mitral stenosis, or what is known as "button-hole" mitral. This lesion is less uncommon in England and Scotland, where the reports of autopsies seem to indicate a greater prevalence of rheumatism than in America.

In speaking to our pathologist of the hospital, Dr. Cattell, he stated that he had not seen more than ten or twelve instances of this condition, notwithstanding the fact that he had made over two thousand post-mortems under the most varied conditions. In reading Fagge's work, I find that he reports that sixty-seven cases were found by him at Guy's Hospital in three years. The degree of the stenosis, however, is not stated. Making allowance for the fact that Guy's is a much larger hospital than this, that number is much in excess of what we find here, and justifies the conclusion that the condition is much more common than with us.

This, then, is a typical "button-hole" mitral. When the mitral valve is diseased in this way, the process begins at the apex of the valve, and not at its base. A curious and striking fact noticed when the specimen is examined is that the left auricle is not materially dilated or hypertrophied. The majority of text-books teach that this is the condition present in well-marked cases of mitral stenosis. My experience is against this, as I have seen marked cases of stenosis in which dilatation of the auricle was not present, and in this instance it is not nearly in proportion to the changes in the ventricle. From the history of the case we may infer that this has been an old, long-continued lesion which would be favorable for such dilatation were it a necessary accompaniment.

The normal mitral valve should allow the passage of two fingers, and then their slight separation. Fagge says three fingers should be admitted, but I have not found this to be the case. However this may be in the normal heart, the mitral valve in this specimen is so contracted that the index-finger is admitted only to between the first and second joints. An interesting experiment, and one on which pathologists do not lay enough stress, is the hydrostatic test of filling the ventricle with water from a tap or

syringe introduced through the aorta and observing its action upon the valve. I tried this yesterday with this heart, and found that there was not only stenosis here, but that the valve also leaked, showing that regurgitation was present. The anterior flap of the mitral valve is the freer, and does the greater part of the work of the valve when in health. It is interesting to note that this is also the case in this greatly diseased valve, as shown by the relative movability of the leaflets.

The kidneys from this case are fibroid and larger than normal organs. There is no doubt that a great deal of the true kidney substance has been destroyed and its place taken by a round-cell infiltration. This is called a fibroid kidney, but the amount of fibroid tissue visible under the microscope in such cases is small, the round-cell infiltration being the conspicuous element, and the two taking the place of true kidney substance. The cortex of the organ is distinctly reduced in thickness. In cases like that of this woman, in which the lung involvement finally caused death, there is an intense congestion of all of the internal organs; and in this case the kidney doubtless became swollen during the last few weeks of life, which accounts for its present enlarged condition.

The liver is roughened, particularly at the lower portion, and more or less hob-nailed over its entire surface. No doubt there is a great deal of fibroid tissue throughout the organ, as is the case with the kidney. The organ is distinctly lighter in color than is normal.

The spleen is in the same condition as the organs just mentioned, being small, its surface uneven, and the capsule showing small white areas of fibroid deposit. That there is considerable fibrosis is shown by the fact that the organ is much harder than normal.

So we have here a case of mitral stenosis, with fibrosis and acute congestion of the kidneys, fibrosis of the liver and spleen, and in addition, a condition most serious of all at the last, the congestion of the lungs. It is this complication which brings about death in the great majority of these cases. The heart is diseased and the kidneys are diseased, while the liver and spleen are also involved, but the lung lesion is the immediate cause of death. In general we may say that death results from one of three causes,—failure of the nervous system, failure of the heart, or failure of the lungs. Laennec spoke

of these as the "three-legged stool of life." I do not mean to say that there are not underlying diseases that cause death, but it is their influence upon one of these three organs that determines the fatal issue. Death comes most rapidly from injury to the nervous system, or its failure to work. Next is heart-failure. A deer shot through the heart may go for some little distance before falling dead, and the same is true of the heart in disease; but death from this cause is comparatively sudden. Slowest of all in causing death is failure of the lungs; but at the same time it is the most frequent cause. This was plainly shown by the congestion and fluid in the pleural sac when the patient was first in the hospital, and her cyanotic and moribund condition when she entered the last time.

One thing more in regard to the mitral stenosis. Do not be too much discouraged if you fail to diagnose accurately this condition. There is really not much difference to the patient whether there is stenosis, or regurgitation, or a dilated heart, or all three, as is the case in this specimen. They all react on the lungs and so cause death. I have so often been wrong, or at least unwilling to express a positive opinion in these cases, that I have less faith in my ability to diagnose them than formerly. Perhaps it is easier for English and Scotch physicians because of their larger experience with cardiac lesions of this kind to be more positive in their diagnosis of mitral stenosis.

Case II.—In this case there has also been difficulty as regards the diagnosis, but of this we now have a pretty good understanding.

The patient is forty-five years old, and was admitted to the surgical ward of the hospital December 27, with the history of having fallen and injured her ankle.

At first a fracture was believed to be present, but the injury proved to be only a sprain. The woman seemed to be considerably under the influence of liquor when she was brought to the hospital. An examination of the urine showed it to contain albumen and a trace of pus, and to have a specific gravity of 1012. The patient's temperature on admission was between 102° and 103° F. Later the respirations increased to 35 or 40 per minute, and there was some roughening of the respiratory murmur. The symptoms became worse, and it was thought that some disease of the lungs was developing, so she was transferred to the medical ward January 1.

The patient, then, came to the hospital suffering from an injury

and slight alcoholism, and was removed to the medical ward with some lung affection developing.

On January 1 her temperature was the same as when she was admitted,—between 102° and 103° F. In a few days it fell, and of late it has been subnormal.

The patient was under observation from January 1 until January 9 before satisfactory evidence of pulmonary disease was found. On the latter date there were marked dulness over the lower part of the right lung and almost entire absence of breath-sounds. An adventitious sound was heard, which I characterized as "crumpling" in character. Examination at present shows but little difference on percussion posteriorly. There is decided harshness of respiration over the lower part of the right lung.

In regard to the sound which I describe as "crumpling," some would say it indicated the presence of pleurisy. In my opinion, the crepitant râle and pleural friction sound are so nearly alike that often the difference between them cannot be made out. Even Skoda, with his refined powers of diagnosis, was not sure of this point. There is probably some pleurisy here, however, though we cannot be positive of its existence.

We had then a curious condition in this patient, and an interesting one. This class of cases often gives the same history. That is, that drinking-people are taken sick when an accident occurs and develop pneumonia, etc. This woman also had albumen in the urine and was more sick than the condition of the lung would warrant. Hence we feel sure that there is some disease of the kidney as well as that of the lung which has been precipitated by the accident.

There was some doubt as to the prognosis at one time, as the patient's tongue was dry as in typhoid fever, and she was violently delirious, but she is now a great deal better.

I stated that it took eight days for positive physical signs to develop, and the patient was worse before they appeared. It sometimes happens this way, and the patient may be really recovering when the definite signs appear.

The treatment has been as follows: five minims of the tincture of digitalis were given four times a day; in addition two and one-half grains of ammonium carbonate and twenty minims of paregoric were administered every two hours.

Another point was this: when the patient was violently delirious I ordered the resident to give her fifteen drops of laudanum at night; and if that did not quiet her, ten drops more were to be given in one hour. If that had no effect, no more was to be given. This gave very satisfactory results in controlling the patient, and will frequently be found of great service.

It seems that opium is not a very popular drug at the present time, but I see no danger in giving it in this way. I have never seen harm from its use, and often the most happy effect. I do not believe in big doses. If a moderate dose does not produce the desired effect in these instances, large doses will not.

Case III.—The patient I now bring before you is a colored woman of thirty-five, admitted to the hospital two weeks ago. Her father died of heart disease. The patient's history is that she has usually been strong and healthy, with the exception of an attack of small-pox when she was a child and a right-sided pleurisy thirteen years ago.

In April, 1899, she contracted a severe cold, this being accompanied by cough and blood-stained expectoration for some time. She also had pain in the chest and abdomen, and soon after her feet began to swell during the day. Following this her abdomen became distended and marked dyspnœa developed. For the last three weeks she was gradually getting worse, and finally started to walk to the hospital, but, becoming exhausted, she was brought in by a patrol wagon.

The temperature has not followed any fixed rule, being 99° at one time, then 100°, then 97° F. The patient had an enormous dropsy when she came to the hospital, and enough remains to make pitting easily seen on the hands. The œdema of the legs was marked, but is less now than of the arms. The skin of the legs has been irritated, as is the case in many instances of this condition, and is puckered and hard. The abdomen was very much swollen when the patient came to the hospital. It is less so now, but ascites is present and the abdomen is tender. Examination of the urine shows it to contain albumen but no casts.

The patient is in a half-sitting posture in bed because of dyspnœa, and physical examination shows marked dulness over the right lung posteriorly, and weakened respiratory sounds. The heart shows no marked heaving impulse, but the apex-beat is below

and to the outer side of its natural position, and there is an increase in the force of its beat. A systolic murmur was heard at the apex some time ago, and this we find to be still present, with reduplication of the second sound. There is an absence of increased percussion dulness of the heart, but this is no evidence that the organ is not increased in size, as the lung is generally emphysematous in these cases. In the first case, if there had not been the stenosis found post mortem, there would have been doubt as to whether it should be called Bright's disease or organic disease of the heart. What have we in this case? The history is that eight months ago the patient was seized with a cough; bloody expectoration was noticed; after a while the feet began swelling, dyspnœa followed, and ascites developed. The patient kept on working these eight months, finally started for the hospital, fell in the street, and was brought in by the patrol.

It used to be that if loud murmurs were found in such cases, they were called organic heart disease; if albumen and casts were present in the urine, they were diagnosed as Bright's disease. But in the course of time cases like this were found in which the disease seemed to be only in the lungs for a time. I have tried to determine whether the disease began in the heart or the kidneys, but do not know. There was disease of the heart, lungs, spleen, liver, and kidneys in the other case, and there is probably the same condition here, but there was no doubt as to which of these was first. There is no name for this when the disease is found in the lungs, and we shall probably put it down as a case of organic heart disease because there are no casts in the urine. As in the other case, the lungs will likely be the cause of death, if the patient does not recover, unless, as sometimes happens, the heart is so rotten that it suddenly gives out. Rotten is the best word to describe this condition of the heart muscle. If this is not present, and we can get the congestion of the lungs relieved, the woman may become better and be able to go out of the hospital for a while as the other woman did. She is improving, but her mind wanders at times. This is not a favorable symptom, as often these patients do not get well when nervous symptoms are manifested.

What are we to do for this woman in order to enable her to leave the hospital?

First, she is to be placed in bed and kept there. When I was a

resident physician here such patients were allowed to sit up or hang over a chair, to help the accessory muscles of respiration. This is not allowed any more. Just what has brought about the change is hard to say, but I think it is best to ascribe it to an evolution regarding the art of nursing. We also used to say that we did not care about the diet of such patients; but this has also changed, and now the two important things are absolute rest in bed and a liquid diet. It is a fact that a much larger number of these patients get on their feet and finally go out of the hospital relieved of their acute symptoms now than formerly, and I think that the two points just mentioned have much to do with this change. As to drugs, we have the choice of several in these cases,—digitalis, strychnine, whiskey, ammonium carbonate. This patient was given ten minims of the tincture of digitalis every four hours at first, this being continued two or three days. A point to remember in the use of digitalis is that it can be given in large doses if this is done for only three or four days and not continued. The dose can then be diminished and continued as long as desired. After having given it in this way, we reduced the amount to five minims three times a day, and this dose can be given for years if necessary with no danger from its cumulative effect. Two and one-half grains of ammonium carbonate are also administered every four hours. Why give a stimulating expectorant in this case? Because the thing the woman is going to die of if not relieved is the congestion of the lungs. One-half ounce of whiskey is also given three times a day.

Often in these cases there is a great amount of pain and oppression, and much relief is obtained by the proper use of opium in some form. In such cases, in addition to the remedies mentioned, one-sixteenth grain of morphine, or one-fourth grain of powdered opium, or five grains of Dover's powder may be given with great advantage.

The physician must have patience in these cases, as anything like a cure of the acute symptoms will take weeks and perhaps months.

The prognosis here is very difficult. The patient will not get well, but she may get a great deal better. The prognosis is rendered more uncertain because of the fact that it is not known in which organ the trouble really began.

I wish to show to-day [1] some specimens taken from the body of Case III, who died in the hospital two days ago (see page 119). Before death the woman was not delirious, but greatly oppressed, and almost screamed for breath. From the description of the symptoms, I thought that she died from clotting of blood in the heart, which opinion was verified by the post-mortem. In this case death could not be referred to the nervous system or the lungs, and, as shown, the final event depended on the condition of the heart.

At the post-mortem we found more than we looked for, and some things that were not recognized during life. As to the lungs, we think they were the parts first stricken in the complication of diseases which this woman had. Though only thirty-five years of age, she had a high degree of emphysema of the left lung. The lung is now soft and soggy, and shows no evidence of a pneumonia. Around the borders may be seen patches like blisters which collapse when cut open, and when pressed between the fingers produce a crackling sound. This shows that they were cut off from the lung and distended because they could not empty themselves through the bronchi. In this condition the capillary supply is practically destroyed, there not being one where there were fifty or even five hundred capillary vessels before, and the walls of the cavities are made up almost wholly of smooth fibrous tissue. This process is almost an essential accompaniment of old age, and in this case the woman was old before her time, as her age and condition indicated.

An examination of the heart shows that the loud mitral systolic murmur heard before death was no evidence of the existence of a valve-lesion, as none exists. Ever since I began studying medicine I have heard that this murmur was indicative of a lesion of the valve; but the fact is that in a majority of the instances in which a mitral systolic or a mitral regurgitant murmur is heard there is no lesion of the valve. This is true here, and it is true in most of the cases that are followed to the post-mortem room. It is nice to tell patients of this lesion, and to congratulate ourselves on the diagnosis; but it is not so nice when we examine the case at the post-mortem and find no lesion. The truth is that generally the mitral orifice is distended by the condition of the heart and the

[1] Clinic of January 17, 1900.

valves do not close. The safety-valve action of the tricuspid valve has been written about; and if this be true of the tricuspid valve, I see no reason why it should not be true of the mitral, although to a less degree because of its different anatomical structure. The ring of tissue surrounding the tricuspid valve is all muscular; that around the mitral valve is one-third fibrous and two-thirds muscular, and this allows of less dilatation of the ring and consequent separation of the valve-cusps. I believe that murmurs of the kind mentioned are often due to the leakage caused by this distention. They may cease at any time when the condition giving rise to the distention disappears. I studied this matter out for myself by experimenting with hydrostatic pressure upon hearts removed at autopsies. The tricuspid valve always leaks when water is forced into the ventricle through the pulmonary artery by a syringe. I inserted a string in the wall of the heart in a circle around the valve far enough from the valve itself not to involve it; when this string was tightened slightly the leakage was less, and when the constriction reached a certain degree, the valve did not leak at all. The same is true of the mitral valve. I think this is a demonstration that a mitral murmur is very often due to a stretching of the heart and valves, and not to actual disease of the valve. The anterior flap of the mitral valve in this specimen is soft and natural, no inflammation being present. The same is true of the posterior flap.

As predicted from the symptoms immediately preceding death, clots in various stages of organization are present in both ventricle and auricle. The aortic valves are not diseased. A much greater amount of blood-clot is in the right ventricle than in the left, which is to be expected, as clots are much more likely to be found in the right heart than the left. The tricuspid valve opening is large, easily admitting my thumb and four fingers.

At the post-mortem in this case we found a curious condition which showed that the patient really died of pelvic disease. I spoke of the bladder symptoms. I had a competent gynæcologist examine the patient, and no pelvic disease except the greatly thickened bladder could be made out. The post-mortem showed the presence of a pocket of pus low down in one side of the pelvis. There are also a number of small fibroids attached to the uterus and one of some size in the uterine wall. The uterus, however, was

not greatly enlarged and was more freely movable than is ordinarily the case when fibroid disease is present. In addition to this, the woman had a surgical kidney. The kidney presents the same appearance as it would if affected by calculous pyelitis, but there are no calculi present.

There are also cystic cavities filled with cheesy material scattered through the kidney tissue, which suggest the possible existence of tuberculous disease.

The patient has had Bright's disease, disease of the lungs, and involvement of the heart. She was affected at least six months before her death. There may have been pus present, but the Bright's disease became progressively worse; the woman died of the former when she was somewhat better of the Bright's disease.

An interesting point is the complication present in this case, and the fact that while so much was known this was overlooked. This will not be thought strange, however, when I tell you that we spent nearly two hours in the post-mortem room studying the condition of the pelvis, and even then could not determine the true state of affairs in the case. We do not know the sequence of events, we do not know where the process began, we do not know whether the pus came from the Fallopian tubes—from gonorrhœal involvement, etc.,—or from outside the uterus.

I wish to direct attention to the difficulty of diagnosis. A skilled gynæcologist could not discover the presence of pus, and I do not think that any one could. But if we cannot tell the exact condition present after we look at the organs, it is not surprising that it could not be determined during the life of the patient.

I am of the opinion that nothing could have been done to save the life of the woman had the condition been fully known. It would have been impossible to cure the disease of the kidneys. If any operation had been done, I think the patient would have died sooner than she did.

AORTIC AND MITRAL REGURGITATION.

A CLINICAL LECTURE DELIVERED AT THE EAST END DISPENSARY.

BY FRANK A. JONES, M.D.,

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GENTLEMEN,—Of the two patients before you, one, the woman, presents all the symptoms and physical signs of typical mitral regurgitation, the other, the man, has all the characteristic symptoms of aortic regurgitation. I shall endeavor to contrast these two valvular lesions in order that you may thoroughly comprehend them, and be able to institute intelligent treatment, as occasion demands.

The woman's clinical history is vague. She gives no history of having had rheumatism; she says that two years ago she suffered from what the doctor called typho-malarial fever, which confined her to her bed for two weeks. She admits having had syphilis. There are numerous enlargements of the glands of the neck. The patient's health has been good since the attack of fever. About two months since, however, she noticed that undue exercise produced dyspnœa; she says that she has "spells" at night with shortness of breath that oblige her to sit up. You notice that cough and dyspnœa are now well marked. There is considerable œdema in the lower extremities; the patient says that her hands are puffed when she rises in the morning. Examination of her urine shows no albumen. There is no effusion into the pleura, a normal, dull note is elicited on percussion. The respiratory murmur is clear on both sides. Expansion is good.

You will note on inspection that the apex of the heart is but slightly displaced, showing that the left ventricle has not as yet become hypertrophied. Upon auscultation a soft, breezy murmur is detected at the apex; it is also distinctly heard in the axilla, and at the angle of the scapula posteriorly. The murmur is so well defined as completely to mask the first sound of the heart.

The pulse is regular but fast. An irregular pulse in mitral regurgitation is, to a certain degree, pathognomonic,—irregularity, both as to rhythm and force, depends upon the degree of venous engorgement of the right chambers of the heart and of the lungs.

The symptoms and physical signs justify the diagnosis of mitral regurgitation. Acute endocarditis, resulting from acute articular rheumatism, is, as you know, one of the most frequent factors in the production of this, the most common of all valvular lesions. But the patient gives no history of rheumatism. Possibly the attack of fever she mentions may have produced an endocarditis, upon which the present mitral lesion depends. Syphilis is so frequent in the negro that it must not be overlooked as a possible cause of endocarditis, from which permanent valvular lesions may arise.

Just how long the murmur has existed we cannot say. Often in the clinic we find mitral regurgitation present when there are no symptoms which point to its existence. Just so long as the muscular tone of the heart is unimpaired, and the right ventricle compensates for the valvular deficiency, there will be no symptoms. But so soon as degenerative changes manifest themselves in the muscular structure of the heart, the hypertrophied ventricle can no longer compensate for the valvular defect, and dilatation begins. Then follows the chain of symptoms which are present in the patient before you.

You will note that there are dyspnœa, cough, dropsy of the lower extremities,—all classical signs of mitral regurgitation with failing compensation. How are we to explain these symptoms? Their etiology may be summed up in these words: Passive congestion, venous stasis. With each systole a stream of blood is thrown from the left ventricle back into the left auricle through the mitral opening. At the same time the normal flow of blood from the lungs into the auricle takes place through the pulmonary veins. In this way the left auricle becomes surcharged with blood from the abnormal current backward through the incompetent valve, and from the normal current from the systemic circulation. When the right and left ventricles can no longer come to the aid of the left auricle—when they have become exhausted, so to speak, and dilated by their efforts to relieve the auricle of this extra amount of blood—backward pressure is exerted through the auricle upon

the pulmonary veins; this pressure in the pulmonary veins spends its force upon the lungs; hence we have dyspnœa as a result of the engorgement of these organs; also cough from irritation of the terminal bronchial tubes. The lungs being thus engorged and congested produce venous obstruction in the right chambers of the heart. The right ventricle is overtaxed in propelling the normal venous blood of the venæ cavæ coming from the right auricle through the lungs, already engorged and congested from backward pressure through the pulmonary veins. The overdistended right ventricle becomes dilated. The venous blood is then forced back from the right ventricle into the right auricle. The right auricle in turns resists the backward flow from the ventricle, because, being the venous reservoir which receives all the systemic venous blood through the venæ cavæ, it cannot perform its function as a reservoir and sustain this backward column of blood at the same time.

There is stasis in the venæ cavæ, which is followed by transudation of serum into the connective tissues—dropsy. Nearly all the organs of the body suffer from this general stasis, more particularly the lungs, the right heart, the liver, and the kidneys. Dropsy in mitral regurgitation signifies failing compensation,—the extent of the dropsy depending of course upon the degree of venous stasis, the state of the walls of the blood-vessels, and the watery condition of the blood.

I wish now to call your attention to this patient, the man, who has another quite common valvular lesion—aortic regurgitation. In its manifestations and physical signs it is the opposite in every respect to mitral regurgitation.

He gives a clear history. Six years ago he had syphilis and underwent treatment for it. Two years since, while attempting to lift a heavy trunk, he lost his balance and fell; upon arising he states that he had agonizing pain in chest, and felt as if something had broken. He comes to-day to consult us for "stabbing pains in the chest" and for a sense of oppression experienced in attempting to do manual work.

Upon inspection the apex is seen to be markedly displaced downward and to the left in the sixth intercostal space; and the apex-beat is tumultuous and bounding. Watch the pulsation in the carotid and temporal arteries; you note that these vessels are

plainly visible, and that they lift my palpating finger with considerable force. I now lift the patient's arms; you can see pulsation from the wrist to the axilla. Upon auscultation a loud, harsh, almost musical murmur, diastolic in time, is heard at the base in the aortic interspace, and is transmitted down to the sternum. We may exclude aneurism of the arch of aorta, as there is no hoarseness, no pulsation nor tumor to right of sternum in the third interspace, the site of these signs in aneurism of the arch of aorta. Neither is there inequality of pulse nor tracheal tugging.

The physical signs justify the diagnosis of aortic regurgitation. These are, first, hypertrophy of the left ventricle, with tumultuous apex-beat; second, throbbing pulsation in the vessels of the neck and head, especially in the carotids; third, a harsh, almost musical diastolic murmur at the base, transmitted down the sternum; fourth, a hard, bounding, radial pulse—the Corrigan pulse.

Aortic regurgitation is not as apt to follow endocarditis as is mitral regurgitation. Syphilis, alcoholism, and muscular strain are frequent exciting causes. This man gives a history of syphilis; also of muscular strain, which, I think, has produced the valvular defect.

Aortic regurgitation is frequently accompanied by arterio-sclerosis,—a fibrous overgrowth of the inner and middle coats of the arteries. It is well marked in this patient. In nearly all the cases of typical aortic regurgitation which have come under my observation this condition of the vessels has been present.

Both aortic insufficiency and arterio-sclerosis are found in late adult life. If either occurs before the age of twenty-five, look for syphilis or alcoholism.

You will observe that this man has neither dropsy, dyspnœa, nor cough,—symptoms always present in mitral regurgitation when compensation is failing. If dropsy develops when the physical signs point to aortic regurgitation, you may look for relative mitral insufficiency with regurgitation as a result of dilatation and degenerative changes in the overhypertrophied left ventricle; in other words, dropsy is not a symptom of uncomplicated aortic regurgitation. The left ventricle is taxed to its utmost capacity in aortic regurgitation for two reasons: First, to compensate for the valvular defect. The aortic valve failing to close with dias-

tole, an abnormal column of blood is forced back into the ventricle. At the same time, with each diastole the normal current has passed from the auricle through the mitral opening into the ventricle; the ventricle, therefore, has to hypertrophy in order to propel this extra amount of blood. Second, vascular resistance resulting from arteriosclerosis must also be overcome. This fibrous overgrowth in the inner coat of the arteries destroys their elasticity and renders them hard and brittle. The function of the elastic coat is to assist the heart in distributing blood to the tissues; if it is destroyed, all the work falls upon the heart, and hypertrophy necessarily follows.

You see how little mitral regurgitation and aortic regurgitation have in common.

After you have learned to recognize the valvular lesions by careful clinical study, their treatment must be considered. *Routinism is the crying evil of the day in treating these affections.* Intelligent application of therapeutic measures for the relief of heart lesions is imperative.

Now, how are these two patients to be treated? One has mitral and the other aortic regurgitation. Digitalis is clearly indicated in the former, but will do positive harm in the latter.

What are the indications for digitalis? Dropsy, stasis, engorgement, lack of muscular tone. We will give this woman digitalis for these conditions. It will enable the right ventricle to contract with more vigor and propel the blood through the lungs, thus relieving their engorgement and the respiratory distress. We give it as a heart tonic, to tone up the muscular structure of both ventricles and cause them to contract more powerfully. The right chambers of the heart also will thereby be relieved to a great extent of venous engorgement, while the left chambers at the same time will be enabled to throw more arterial blood into the general system. By securing a better supply of arterial blood, neutralizing the valvular defect, so to speak, and by relieving the engorgement in the right chambers of the heart and the consequent venous stasis, the cause of the dropsy is removed. Hence the value of digitalis in mitral regurgitation. Free purgation in conjunction with digitalis is always imperative when dyspnœa and dropsy are present. I have found no better agent than calomel; it relieves the engorgement of the liver, and also that of the kidneys. We will purge this

patient once or twice a week with ten-grain doses of calomel; we will give her minute doses of bichloride of mercury with digitalis three times a day, and watch her progress. We have not time to touch upon diet to-day, except to say that easily digested liquid diet is indicated; sweet milk will be given in abundance.

We now again call your attention to the man who has aortic regurgitation. We shall certainly not give him digitalis. To do so, with the conditions confronting us, would be positively injurious. His arteries are constricted, hard, unyielding, as a result of arteriosclerosis, which is well marked. This condition offers resistance to the distribution of blood to the tissues. Digitalis, acting as a vaso-constrictor, would increase the already high blood tension and materially aggravate his symptoms. Even if he had no arteriosclerosis with this bounding apex beat, this throbbing pulsation in the carotids, and this hard Corrigan pulse, digitalis would be of no avail. My experience has taught me that *digitalis is never indicated in aortic regurgitation unless dropsy manifests itself as a result of relative mitral regurgitation.* Even then I doubt its efficacy. Broadbent, whom I regard as the best living authority on heart disease, says, "In the absence of mitral symptoms it is rarely that digitalis is called for in aortic incompetence, or is of service, and it may undoubtedly do harm. It may set up sickness, which is an ominous symptom in this form of disease, and induce a condition of asthenia difficult to remedy." This man should be put to bed. Rest is more essential than medicine. We will give him two-drop doses of aconite three times daily to quiet the tumultuous heart-action. We will also give him a tonic of arsenic and nitroglycerin; the arsenic for its general tonic effects; the nitroglycerin as a vaso-dilator. The blood-vessels are constricted; the vascular tension is high. Nitroglycerin (or strychnine) overcomes the constriction, the blood-vessels dilate, and the diminished tension thus secured relieves the demand upon the hypertrophied ventricles.

A SERVICE IN THE MEDICAL OUT-PATIENT DEPARTMENT AT THE PENNSYLVANIA HOSPITAL.

BY HARVEY SHOEMAKER, M.D., AND EDWARD LODHOLZ, M.D.,
Of Philadelphia.

The following *résumé* of the cases treated at the out-patient department of the Pennsylvania Hospital during the months of June, July, and August, 1900, has been prepared from the notes taken at the clinic. No attempt has been made to consider any case or subject in detail, but only to give the general character of the service at this hospital, and to outline the treatment adopted in those minor affections which form so large a part of the routine practice of a physician.

THE DYSPEPSIAS (INDIGESTIONS).

Of the total number of patients treated at this dispensary over seventy per cent. are of foreign birth and almost fifty per cent. are Russian Jews. Living in the most unhygienic surroundings, and subsisting on food of the poorest quality, which they take at irregular intervals and in most varying quantities, it is surprising that but fourteen per cent. were cases of primary indigestion. One of the symptoms most frequently complained of was flatulence, which occurred in nearly one-half of the cases treated. By its indirect action on the heart it not rarely produced præcordial distress with palpitation, directing the patient's attention to the heart and causing considerable alarm. Epigastric distress and tenderness were present in fifty of the one hundred and three cases, and there was headache in twenty-five cases. Constipation coexisted in over one-half of the total number.

Treatment.—Having neither the time nor a sufficient number of assistants to use the stomach-tube, and in this way to make accurate diagnoses by chemical analysis, the symptoms furnished the indications for treatment. Patients with flatulence, acid eructations, sensation of fulness, constipation, and more or less dilatation of the stomach, were often treated with sodium bicarbonate and

fluid extract of cascara combined with peppermint water. This preparation was used in thirty-six cases; twenty-one did not return, in fifteen considerable improvement was noticed, and in two cases of gastralgia it seemed to intensify the symptoms.

When marked anorexia existed and the other symptoms were present only in a mild degree, the bitters preferred were tincture of nux vomica, alone or combined with gentian, usually in the form of the infusion. Thirteen patients were so treated; six were much improved and the others made no return visits. If combined with anorexia, and there was distress for a while after eating, with little or no flatulence, no acid eructations, and no constipation, dilute hydrochloric acid and pepsin were added to the bitter infusion. Three of the six cases so treated returned improved. Dilute nitrohydrochloric acid was substituted for the hydrochloric acid when the liver was thought to be involved, as evinced by sallowness, slight enlargement, etc.

In cases of long standing, particularly when other drugs had failed, and in cases of gastralgia nitrate of silver was given in pill combined with kaolin and petrolatum a half-hour or more before meals. The kaolin was used to prevent oxidation of the silver. Of the nine cases four improved under its administration. Its influence was most decided in two cases of gastralgia, where other remedies had failed.

In acute cases, and particularly where the inflammation had extended to the intestines and produced diarrhœa, calomel was given in small doses frequently repeated, followed usually by a powder of bismuth salicylate or subnitrate and pepsin. Twenty-eight patients received this treatment, and the seven who remained under observation either improved or recovered entirely.

Laxatives were prescribed for continued use only when attention to diet, etc., had been resorted to without effect. Preference was given to sodium phosphate in one- to three-drachm doses in hot water before breakfast. When very prolonged laxative action was necessary, pills of aloin, belladonna, and strychnine were used.

RHEUMATISM.

The most unsatisfactory cases to treat in dispensary practice are those that are diagnosed rheumatism. There is always an appreciable percentage of malingerers who simulate the chronic form

of the disease for the purpose of obtaining either admission to the hospital or a signature to a certificate for a sick benefit. The simulation is often so perfect, since the leading questions of the physicians who previously examined the patients have taught them so much, that positive deductions cannot be made. Again, there is a second class of cases giving symptoms so vague that it is not possible in so short a time to arrive at the true nature of the disease. Thus, there were eighty-seven cases diagnosed rheumatism, and yet fifteen more would have been so called had not further investigation proved them to be cases of neuritis. Then, again, closely resembling it were two cases of phlebitis involving the deep femoral vessels. Tenderness along these vessels was previously thought to be due to the sciatic and anterior crural nerves, even though there were cyanosis and swelling of the lower part of the limbs. The subjective symptoms in both cases were exactly like those of rheumatism. Swelling, redness, and pain of the wrist on two occasions were distinguished from the ordinary rheumatic affection of that joint by the grating produced over the tendons on extension and flexion. Another patient with all the symptoms of subacute rheumatism had typical lesions of erythema nodosum on the legs. Of all the patients treated for this disease but seventeen had joint involvement. Nearly all at first complained of pain and tenderness and weakness in a joint, but examination showed that the muscles or other structures near the joint were the parts really at fault. Swelling of the joints was even more rare, occurring in but ten cases. Rheumatic fever was seen but once. The structures most commonly involved were the muscles. The erectores spinæ in the lumbar region were the seat of trouble in one-fourth and the muscles of the chest in one-eighth of the cases. When these muscles were affected, usually diagnosis by exclusion was resorted to. A patient would complain of pain of very sudden development in the right or less frequently in the left chest. This pain was constant and always intensified by deep inspiration. It would continue with varying intensity for a week or two yet constitutional symptoms would practically be absent. Repeated examinations when possible would detect no friction sound nor could there be elicited local tenderness.

The muscles of the neck were affected but twice; in both instances the rigidity and pain on motion greatly exceeded that which existed when any other muscle was involved.

The sheath of the tendons was the seat of trouble twice. One case was undoubtedly rheumatic, the other doubtfully so.

The study of the etiology was usually indefinite. Of cases in which the exciting cause could be learned, six were due to sore throat, three attributed to exposure to cold and wet, and two to syphilis.

Constitutional symptoms were usually absent; when present the most marked were headache, constipation, and anorexia; strange to say, heart murmur was present but seven times.

Acute cases were treated with salicylates and chronic cases with potassium iodide. Salicylates were prescribed for fifty-one patients, the sodium salt being given unless there was weakness of the heart, when ammonium salicylate was used; where there was irritability of the stomach the strontium salt was preferred. Of the fifteen patients that returned fourteen were improved. The results obtained from potassium iodide were disappointing. It was tried fourteen times; but two patients returned, one improved and the other no better. The two drugs were combined in five cases; one returned improved.

Often local treatment was added, in the hope of obtaining good results from the massage necessary in its application, preference being given to soap liniment. This was frequently combined with aconite or oil of gaultheria. Ichthyol, fifty per cent., was applied when there were marked joint symptoms. The results obtained from these various local applications could not be definitely estimated, as internal remedies were always given at the same time.

NEURITIS.

In but two cases were the nerves of the arms independently implicated; in one the cause was lead-poisoning, and in the other, pressure due to the position of the patient when under the influence of alcohol. In both instances there was decided wrist-drop. The nerves of the chest were affected three times, and in every instance the typical blebs of herpes zoster following the course of the nerve-trunks were seen. Pain in the chest of a severe type previous to the development of the skin manifestation existed in two of the cases.

In three cases in which the facial nerve was involved the inflammation seemed to be located at the exit of the stylomastoid foramen.

Coming on insidiously the first symptom noticed was difficulty in chewing. Examination showed freedom from involvement of the sense of taste; the uvula was in its normal position; there was no disturbance of hearing; but the inability to pucker the lips and to close the eye and the absence of expression on the affected side were noted. Considerable improvement followed the use of electricity applied at forty-eight-hour intervals. Sciatica was seen four times; it presented nothing worthy of note except that drugs had no influence whatever.

Œdema of the feet and severe pains in the extremities, associated with greatly exaggerated reflexes and extreme tenderness over all the nerves, in a patient convalescing from typhoid fever, led to the diagnosis of multiple neuritis. The cases of polyneuritis were particularly interesting because on first observation they looked not unlike chronic rheumatism. The symptoms in the ten cases were pains, rheumatic in character, in the arms, chest, legs, or body, with loss of power. Examination of the affected part revealed tenderness over the nerve-trunks, with at times considerable wasting and exaggerated reflexes. Constitutional symptoms were absent. Gonorrhœa seemed to be the causative factor in all but three instances.

Treatment.—The salicylates seemed to relieve the pain, but did not improve the local condition. Arsenic was given without benefit, also potassium iodide. Iodide of iron was tried in ascending doses twice, but without any manifest benefit.

NEURASTHENIA.

Twenty-three cases of functional nervous disease were treated. In most of these there was either a syphilitic or alcoholic taint. Others were the result of some derangement of the sexual apparatus. Frequently masturbation precipitated the condition. There were, however, some primary cases. The symptoms were of the most varied character—in no two cases alike—and were not associated with any marked organic lesion. The treatment was absolutely unsatisfactory.

TONSILLITIS.

This disease was most unsatisfactory to study,—first, because of the absence of bacteriological evidence, secondly, because of the

very early stage of the disease, and, lastly, on account of the few that returned after the first visit. Of the twenty-nine cases, twelve were seen on the second day of the disease, too early for positive diagnosis, and but four returned for further treatment. Besides the swelling and redness of the tonsils observed in every case, pseudo-membrane was seen six times. The glands at the angle of the jaw were decidedly enlarged in six cases, and once suppuration occurred. Constitutional symptoms were usually acute; high fever, rapid pulse, anorexia, and stiffness and pain in the muscles were the rule. One patient came with the diagnosis of pulmonary tuberculosis, on account of the copious spitting of blood. On examination this was found to be due to very much enlarged and engorged tonsils, which readily yielded to treatment.

Closely resembling tonsillitis was a case seen on the second day of the disease, with enlarged and red tonsils but no membrane. There were marked constitutional symptoms,—fever, rapid pulse, and vomiting. An irritating discharge from a purulent rhinitis, which produced excoriation of the skin around the nasal orifices, led to the diagnosis of diphtheria. Next day this was confirmed, for a gray membrane covered the tonsils and palate and even extended to the roof of the mouth.

The drugs usually given were potassium chlorate or tincture of chloride of iron. When, however, there was high fever, associated with severe headaches, muscular and joint pains throughout the body, acid sweats, and (as there was in one case) swelling of the joints, the salicylates were given.

BRONCHITIS.

Of the total number of cases, thirty-seven, but four were chronic. This form in children was apt to follow measles, whereas no small percentage of the acute cases occurred at the onset of influenza, measles, and pertussis.

Treatment.—In the acute cases, early in the disease and particularly where there was much fever, citrate of potassium was relied upon. It was given nine times. Less frequently syrup of ipecacuanha or squills was added to the mixture. In acute but particularly in the chronic cases where there was considerable irritability of the stomach, heroin was used. In one of the five cases in which it was administered it had a very beneficial action and in

another instance the patient quickly recovered. Neither was irritability of the stomach a contraindication for the use of ammonium chloride. Here it was combined with drugs directed to the stomach, and other expectorants were avoided. It was given to thirteen patients; in three improvement was noticed. In the chronic form preference was given to creosote in ascending doses, using milk as a menstruum, or to terebene in five-drop doses on sugar. Cod-liver oil and malt were also sometimes used. The adjuncts were potassium bromide, when the severity of the cough was out of proportion to the local disease, and sometimes opium. Codeine was not used, on account of its expense. Belladonna was sometimes added to the mixture, as was also compound mixture of glycyrrhiza.

PHTHISIS PULMONALIS.

A patient entering the dispensary complaining of symptoms of dyspepsia, with its associated weaknesses and possibly some vague chest pains, was invariably examined for pulmonary involvement, which in a surprising number of cases was present, even when no cough existed. Twenty-seven cases of pulmonary tuberculosis were treated; symptoms had existed from three weeks to ten years. Gastric derangement, with loss of weight, night-sweats, pains in the chest, weakness, and headache, were mostly complained of. Combined with these were more or less cough and expectoration. Both lungs were involved in four cases, the right apex in sixteen, the right apex only posteriorly in three, and the left apex in four cases. The relation between the pulmonary and the aortic second-sound was carefully studied, and the conclusion arrived at that there was no definite relation.

These patients are most prone to seek medical advice at every accessible dispensary and to take great quantities and varieties of drugs, but to persist in no single course of treatment for any length of time. So of all the cases treated but five returned. The treatment attempted was constitutional and local. Thus, for the general emaciation and weakness, oil-rubs, elixir of quinine, iron, and strychnine, Blaud's pills, and syrup of iodide of iron were given, with, where possible, careful attention to the food and hygiene. For their local action creosote or guaiacol carbonate was administered in ascending doses. Heroin seemed to relieve the cough to some extent.

PNEUMONIA.

The relatively few patients brought to the dispensary with pneumonia are usually children who may be easily carried. Of our six cases four were infants. In one of the adult patients two lobes of one lung were completely consolidated, yet he complained of few symptoms; whereas the other, with less involvement of the lungs, had very severe fever. The children had the catarrhal type of the affection. Two of them lived in the same house and contracted the disease at the same time. Consolidation was extremely difficult to detect. The percussion note was often so slightly altered that the resistance to the percussing finger was of considerably more significance. The breath sounds were sometimes neither bronchial nor broncho-vesicular, but altered just enough to justify the statement that they were not normal.

The treatment was ammonium carbonate in small doses frequently repeated. The more permanent stimulants, such as strychnine and digitalis, were reserved for the latter part of the disease in case of necessity. Cotton jackets and poultices locally to the chest were avoided.

Emphysema followed pneumonia in a patient sixty years old.

PLEURISY

One case of pleuritis was of particular interest. A child seven years old, convalescing from measles, was brought to the dispensary on account of severe pain of twenty days' duration in the lower right side of the chest. There were anorexia, fever, and considerable shortness of breath. Examination of the chest revealed a distinct pleural friction sound at the right base anteriorly, with broncho-vesicular breathing. Two days later the patient returned, and the clinical notes of that date recorded,—"The pain in the side gone; child improved. Some cough persists; some fever; many râles in chest. At right base anteriorly and posteriorly breath-sounds are more feeble, but otherwise not altered. Liver dulness increased." Three days later a soft mass was discovered over the liver. This was subsequently opened and considerable pus removed. The abscess was found to be perihepatic. Another patient had an adherent pleura of the whole right lung, which was easily detected by the almost complete absence of expansion of that side. Pleurisy

with effusion occurred but once. The other cases were seen early in the acute stage.

ENDOCARDITIS.

In the sixteen cases treated it was the exception rather than the rule to get a history of rheumatism, whereas if it could be established positively that sore throat often produces endocarditis every case treated could be accounted for as to causation. One patient had no cardiac symptoms until treated for acute tonsillitis, when an organic endocardial murmur developed. In another instance conditions pointed strongly to tonsillitis as a cause of endocarditis. The most frequent accompanying symptoms were constipation, anorexia, dizziness, slight cough, headache, and weakness.

The lesions were mostly limited to the mitral valves. The aortic valves were not seldom involved, but these cases were generally put under other heads, as other lesions were found that were of more importance than the heart lesion, the most common of which was general sclerosis.

Treatment.—The constipation was first relieved, the appetite was stimulated and tonics were given for the anæmia, and attention was directed to the kidneys, food, and hygiene. In case of failure after fair trial of these procedures, cardiac stimulants were resorted to.

MEASLES.

No rare or abnormal conditions presented themselves. All the patients were seen soon after the appearance of the rash. In every instance this appeared first on the face. The buccal mucous membrane was examined for Koplik's spots, which were found but twice and then only indistinctly. The conjunctiva was also studied with the hope of finding these lesions, but the usual inflammation was all that could be found; catarrhal symptoms were absent twice.

RÖTHELN.

Closely simulating measles were two cases of rötheln. One occurred in a woman aged twenty-four, who had itching and burning sensations in a recently developed rash which involved the whole body. The lesion was macular in most places but erythematous in others. She complained of no constitutional symptoms, but further investigation showed enlarged and inflamed tonsils, swollen lymphatic glands all over the body, and a temperature of 100° F. The

second patient was just recovering from a severe attack of measles; this rash was more erythematous and less macular.

ERYSIPELAS.

An indurated and erythematous swelling of the skin of the face, which was inclined to be wing-shaped on the nose and cheek and accompanied by acute febrile symptoms, made the diagnosis easy. In two cases large dirty blebs covered the lesion. Those affected were in advanced middle life. Convalescence in the cases which returned (four out of ten) was very rapid under large doses of tincture of iron and fifty per cent. ichthyol ointment. There was one recurrence.

GLANDULAR FEVER.

Cases presenting acute enlargement of the cervical glands, with fever and constitutional symptoms and without other lesions, were called glandular fever; yet this diagnosis was almost as frequently revised as it was made. In every case, where there was swelling of the glands without obvious cause, suppuration followed. The pus was examined bacteriologically in one case, and pure cultures of the pneumococcus were isolated. In a second case the throat showed no signs of inflammation. The glands at the angle of the jaw were enlarged. Here an acute coryza was finally considered to have been the cause. The third case was more puzzling, as the source of infection could not be detected. The patient had a slight bronchitis, which suggested the possibility of a catarrhal condition higher up in the respiratory tract at an earlier date. A *résumé* of all the cases treated during the three months' service makes it doubtful whether a single case of true glandular fever presented itself.

DISEASES OF THE SKIN.

Rhus Toxicodendron.—Throughout the month of June not a single case of poisoning by rhus toxicodendron presented itself, but in July there were six and in August ten cases. It occurred almost exclusively among the Italian laborers who were constantly exposed to the plant. The incubation was usually limited to a couple of days. The most common manifestation was an elevated and somewhat indurated erythema on the exposed parts of the body. This was covered with vesicles filled with a clear fluid. When

these were opened by scratching, the parts became covered with a soft, yellowish-brown crust along the course of the scratch mark. Sometimes an erythema with indistinct edges was all that could be seen; at other times the erythema was covered with many large and dirty blebs. All the patients were treated with the fluid extract of grindelia robusta diluted one to thirty-two. In every case that returned improvement was marked. Some lesions were so unlike those usually seen that at times it was thought that another species of the plant must have been the cause.

Impetigo Contagiosa.—The lesions of this disease, with their typical dark-brown crusts having the "stuck on" appearance, were so characteristic that the diagnosis was usually made instantly. These cases appeared frequently, as the source of contagion was wide-spread. Their course was acute. Itching was present twice. One case was of particular interest on account of the great amount of surface affected by the disease: its lesions were found in every part of the body. They started as vesicles (clear); these soon developed into blebs (looking not unlike pemphigus), which finally became purulent and broke down, forming the characteristic crusts. Intense itching coexisted, which was uninfluenced by treatment. The physical condition was excellent and the general health was in no way affected. The cases were treated with one grain of ammoniated mercury to the drachm of simple ointment.

Eczema.—Thirty-four cases of eczema appeared at the clinic; of these six were squamous, two pustular, four intertrigous, two fissural, six papular, one vesicular, and one rubricose; the rest were not differentiated. The squamous variety was interesting etiologically. In most the immoderate use of soap on the face was clearly the cause; once an application of remedial agents to the scrotum for the extermination of pediculi was the cause of the eczema in that part of the body. The pustular form was of interest because in the two patients so affected it involved the hair-follicles of the beard. Here a diagnosis was arrived at only after repeated search for the trichophyton. The intertrigous form twice involved the folds at the back of the ears, which subsequently became covered with a moist, soft, brown exudate. This variety was quite resistant to treatment. The other varieties presented nothing of much interest.

Treatment.—The pasta mollis of Unna succeeded very well.

When there was intense itching, carbolic acid and boracic acid were used in the form of a salve. In all the squamous cases tar was of great benefit; in the chronic intertrigous form calomel one grain to a drachm of ointment seemed to succeed best, and, if acute, zinc and boracic acid were given with benefit. Internal remedies were sometimes used.

Tinea Versicolor.—As there are no subjective symptoms, the disease was discovered only incidentally, as when the chest was exposed for lung examination. Usually the brown lesion with its fine scales had not even attracted the attention of the patient. In the two cases treated the whole anterior and posterior chest was involved. The microsporon furfur and the spores were present.

Tinea Tonsurans.—This was noted three times. The falling of the hair of the involved area, with fine scaling, without subjective symptoms, suggested a microscopic examination, and in every case the trichophyton was found. Improvement followed the use of ammoniated mercury; when this irritated, the yellow oxide was applied.

Tinea Circinata.—Three cases presented themselves. Rapid recovery followed the use of sulphur ointment.

Tinea Sycosis.—The one case looked like epithelioma and had been so diagnosed.

Tinea Favosa.—Once the fungus of this disease was found in a cup-shaped lesion on the head, which was covered with crusts; it improved under the use of tincture of iodine.

Scabies.—In not a single case did the microscope confirm this diagnosis.

Pediculi.—In pediculosis corporis the skin lesion in many instances looked not unlike scabies except that the burrows were absent. Usually there were on the body papules with their tops scratched off, leaving a bloody, intensely itching surface. Careful search of the clothing was necessary to find the pediculi.

Pediculosis Capitis.—The irritation produced by the pediculi and the consequent scratching offer a good opportunity for infection, and as a consequence there frequently developed a red granular base covered with matted hair and a soft yellow crust. Enlarged cervical glands were often seen in these cases at or near the hairy limits of the scalp.

OBSERVATIONS ON THE PHLEBITIS OF ADVANCED PHTHISIS.

BY ROLAND G. CURTIN, M.D.

In the course of a practice extending over a third of a century I have seen a number of cases of phthisis with a complication which, so far as I can find, seems almost to have escaped observation. In sixteen cases I have noticed during the early or middle part of the last stage of acute or subacute phthisis more or less well-marked phlebitis of the veins of the thighs and legs. The symptoms did not tally with those of the swelling of the legs which immediately precedes death, but took place much earlier and then subsided. The condition was a separate and distinct one, of acute, inflammatory origin, and was followed later by the swelling of the lower extremities which usually occurs just before dissolution.

The first case that I saw was that of a girl aged eighteen years, with acute pulmonary tuberculosis, running its course in about four or five months. She had a marked increase of temperature, —two degrees above that which she had had previously,—with a dry skin, increased frequency of the pulse, thirst, a dry tongue, and tenderness along one femoral vein, which later extended down the limb to the ankle. After the symptoms in this leg had run their course and subsided, the phlebitis appeared in the other leg, and later passed away in the same manner. The feverishness decreased, the temperature falling to the degree that had prevailed before the attack; the soreness of the limb subsided, as did also the swelling, until it disappeared entirely. This was about two months before death.

The observations that I have made upon this class of cases all tally more or less closely with those just described, the symptoms differing in severity and the time of occurrence, which was in most

cases weeks or months before the death of the patient. I here place in tabular form the symptoms that I have observed, to show the differential diagnosis between the phlebitis and the œdema of the feet which usually precedes death in cases of pulmonary tuberculosis.

Phlebitis.	Œdema of the Feet at the End of the Last Stage.
Marked increase of fever.	Fever generally not increased.
Marked pain along veins.	No pain; only stiffness below knee.
Marked soreness along veins.	No deep tenderness; skin tender.
Secretions diminished.	Secretions not diminished.
Skin hot.	Skin not hot.
Patient excited and irritable.	Patient feeble and weak.
Swelling decreases in a few days.	Swelling continues to the end.
Symptoms subside.	Symptoms followed by death.
Symptoms usually occur weeks or months before death.	Symptoms occur just before death.
Symptoms generally commence above the knee.	Symptoms begin below the knee.
Generally in the female sex.	In both sexes alike.

It will be seen from this tabulation that the complication to which I call attention is inflammatory, and that it occurs much earlier than the common swelling of the feet which physicians, as well as the laity, regard as a forerunner of death. In this connection the following questions might be raised. (1) Is the swelling of the feet in the latter part of the third stage of phthisis caused by a latent inflammation or by a thrombotic condition of the veins? (2) Are the veins tubercular, becoming involved in the last stage of the disease? (3) Is the swelling caused by the extreme weakness of the circulation? (4) Is it produced by the poor condition of the blood? (5) Is it a stasis of the blood in the veins from interference to the passage of the blood through the diseased lungs? (6) Can it be a combination of poor blood, weak or retarded circulation, and thrombosis?

It has occurred to me that this phlebitic condition might point to a solution of the question. The system, being stronger at an early stage in the disease, is able to react sufficiently to produce the febrile and other symptoms before mentioned. I have failed to find in the literature on the subject of phthisis any description of the phlebitic complication herein described. I have seen physi-

cians and others placed in an awkward position by this complication, they having stated that the swelling indicated the occurrence of death in a few days. The patients recovered from the symptoms, and the prophets were thought to be poor prognosticators.

I have never had an opportunity of ascertaining *post mortem* whether or not the inflammation of the veins was of tubercular origin. Perhaps in these cases there had been a double infection, the phlebitis being the result of the action of the associated bacteria.

Neurology

PROGRESSIVE MUSCULAR ATROPHY.

CLINICAL LESSON DELIVERED IN A SPECIAL COURSE AT THE SALPÊTRIÈRE HOSPITAL IN PARIS.

BY JEAN B. CHARCOT, M.D.,

Sometime Interne of the Hospital of Paris and Chief of the Clinic at the Salpêtrière.

GENTLEMEN,—A number of nervous diseases that only a decade or two ago were considered to be extremely rare have in recent years turned out to be of comparatively frequent occurrence. When these diseases were originally described, many of them were looked upon as great medical curiosities. Since then they have become of distinct practical importance as types of disease that even the general practitioner will occasionally meet. Among these affections is progressive muscular atrophy. It is not so common as syringomyelia or chronic anterior poliomyelitis, but it is of sufficient importance to deserve a clinical lesson in our course on practical nervous diseases.

A good many years ago Duchenne (of Boulogne) and Aran gave a description of a type of progressive muscular atrophy with the following fundamental features: The atrophy develops during adult life. Its beginning is insidious. It commences in the small muscles of the hands and invades slowly but progressively other muscles running from the periphery to the trunk, affecting first the different segments of the upper arms, then the muscles of the trunk, and finally those of the lower limbs. It attacks muscles separately and sometimes affects them only partially, so that it causes progressive enfeeblement. This weakness is in some sort proportional to the lessened volume of the muscles which are affected. The

atrophy is not accompanied by contractures, but fibrillary tremors in the course of the muscles are usually noted. Faradic contractility is preserved for a long time. Usually the muscles of the face are left absolutely unaffected. Only rarely and very tardily does the progressive atrophy attack the muscles of the tongue, of the pharynx, and of the larynx.

When Duchenne first described this type of disease, there were tendencies to group under it a number of ailments that we have since found to be distinct nosological entities. Anatomical research and clinical observation have shown in recent years that various forms of muscular atrophy are due to definite nervous lesions that cause them to be grouped under a number of distinct heads. The fundamental group of muscular atrophy described by Duchenne now occupies a comparatively insignificant place in neurology. It is even a subject of serious discussion in some quarters whether muscular atrophy, as described by Duchenne, exists as an independent affection. It so happens that we have certain cases here at the Salpêtrière at the present moment which illustrate very well the exact type of disease described by Duchenne, and demonstrate that as a clinical entity at least it still holds a very definite place among nervous diseases. After you have seen the patients you will appreciate the truth of this assertion much better.

Our first patient is a wood-worker, forty-three years of age, who has been under observation for some months. The first symptom of his present trouble manifested itself about two years ago. One day, while rolling a cigarette, he noticed that his right thumb was, as he says, "lazy." Up to that time he had never noticed the slightest difficulty in making any movements with his fingers. This disinclination of the thumb to do work to which it was accustomed was but slightly marked. Three or four weeks later he realized that the index-finger of his right hand was suffering from some interference with its movements. Shortly afterwards he was obliged to stop work. In his occupation of wood-worker, as he tells us, it is his right hand which does all the work. In using the chisel, and his occupation requires him to use it for six or eight hours a day, the butt of the tool-handle rests in the hollow of his hand,—as a rule, just between the thenar and hypothenar eminences. The shaft of the steel tool itself is held between his thumb and index-finger, and vigorous action on the part of the

muscles of these two fingers is required to direct it properly. During a day's labor it is almost exclusively the thumb and the index-finger of the right hand which become fatigued. The left hand has only to guide the extremity of the tool and so becomes very little fatigued. Besides this, all the other tools that are used for producing grooves are held in the same way as the chisel, and all of them rest between the thumb and index-finger of the right hand.

Our patient stopped work, then, when he could no longer hold his tools. About six weeks after this he noticed that the movements of the right shoulder were interfered with. Movements of the arm involving the shoulder-joint were accomplished with more difficulty than before, and attempts to use this arm for any serious work were soon followed by a great sense of fatigue.

About three months after he noticed the first symptom in the thumb of his right hand, the thumb of his left hand was affected in the same way. Then his left index-finger became involved. Some weeks later his left shoulder was attacked. The symptomatic process in the left arm followed precisely the same course as that in the right. After the affection of his shoulders, the fingers of both hands gradually grew feebler. About six months since the beginning of his symptoms the patient noticed that his thenar eminence began to decrease in size. Shortly afterwards the first interosseous space became noticeably depressed, from the disappearance of the muscular padding which usually exists there. These symptoms were noted first on the right and then on the left side.

This process of paresis and of muscular atrophy was accomplished without the slightest unusual feeling in the parts affected. There was no sense of numbness, no formication, and absolutely no pain. The patient has only experienced, when each thumb was first affected, a slight sensation of fatigue in the corresponding arm. About six months after the first symptom declared itself, a sensation of extreme feebleness and fatigue, even after slight exertion, came on in the legs. In the morning on getting up he felt tired out,—just as if he had walked fifty miles, he says.

The feebleness of the arms and legs became more accentuated, little by little. After eight months the lower limbs reached the state in which you now see them. For a year and a half since then

the disease has remained stationary in his legs and the feebleness has continued to increase only in his arms. For the last two months the patient has had spasms of fibrillary tremor in the legs, in the calves, and in the lower and upper arm. He has never had any real derangement of his sensibility. During the last few months cramps have occurred sometimes in one and sometimes in the other arm. These cramps do not last long and come on only when the arm has been held for some time in a strained or unnatural position. If the patient changes the position of the arm, the pain disappears.

He does not think that his legs have become emaciated: he is of the opinion that they have always been of about the same size as now. During the last ten days or so the patient has experienced a feeling of painful constraint and discomfort in the cervical region. He has never noticed any feebleness in the movement of his neck or anything abnormal about his face or head.

When he entered the hospital, we searched very carefully into his family history, but it proved to be practically negative. His father died at twenty-six of tuberculosis; his mother is living and well at the age of sixty-two. He has no brothers or sisters. All four of his grandparents were healthy and long-lived. Careful inquiry does not reveal the slightest neurotic strain anywhere in the family. The patient knows the family history well, and is very definite in his denial of any hereditary taint.

His personal history is of the same negative type. He had none of the usual children's diseases. He was at school from four to fourteen years of age, and learned to read, write, and cipher without difficulty. He went through his military service without incident. At the age of eighteen he served for a time as an infantryman in the Franco-German war. He had no venereal diseases, is not given to alcoholism, and has never taken drugs. He is married and has one child, a girl of six years. Some four years ago he had an attack of influenza that lasted for ten days, but recovered from it completely. The following year he had another attack of influenza much severer in character, from which he required a month to convalesce. After that time, however, he felt as well as ever. The following year his muscular atrophy began.

Present Condition.—The patient presents, at first sight, nothing special, except his atrophied hands. The face is normal; the

lips, the tongue, and the velum of the palate are entirely natural. The neck is unaffected. There is no scoliosis and no pain over the spines of the vertebræ when percussion is practised. The patient has no vesico-rectal trouble and no disturbance of his respiratory or circulatory system. The trophic condition of his skin is not at all disturbed. The different viscera are normal and their functions are accomplished without any hinderance. His general condition leaves nothing to desire. Nothing can be found except the disturbed reflexes, certain fibrillary tremors, and the muscular atrophy that has been noticed.

All the tendon reflexes exist, but are diminished. They are more feeble in the right arm and leg than in the left. One might say that they are lessened on the right side and almost normal on the left. In no part of the body are any of the reflexes exaggerated.

The fibrillary twitchings can be noticed more or less everywhere except on the neck and face. They are most marked in the triceps of both arms, in the right deltoid, in the right pectoral, in the triceps of the legs, in the extensors of the forearms, and in the thighs and calves.

The muscular atrophy is quite conspicuous. It causes a feebleness of muscular action proportionate to the absence of muscular tissue. It is more marked in the arms than in the legs, where it would pass unnoticed except by an attentive observer, and it is more distinct on the right than on the left side. The right hand particularly is very much affected. The forearms, however, the upper arms, and the shoulders on both sides are easily seen to be quite atrophic. The muscles of the trunk are very slightly affected. The atrophy that exists in the legs is best brought out by making the patient walk. He becomes fatigued easily and quickly, and from time to time is obliged to stop and rest for a moment. Careful examination of the upper extremity shows that the thumb of the right hand can still be opposed to the middle and index-finger, but not to the ring and little finger. The faculty of opposition in the left thumb is entirely lost. The three outer fingers of the right hand can still be bent into the palm, but this is impossible for the thumb and index finger. In the left hand only the thumb refuses action in the effort to make a fist. His grasp shows that he has very little muscular force. The pressure

can scarcely be felt. The extension of the fingers and their separation and their bringing together are almost impossible.

The hands have not the claw-like appearance so characteristic of extensive atrophy, but the thumbs are on the same plane as the other fingers. The thenar eminences, especially the right, are very distinctly flattened. The atrophy of the hypothenar eminence is much less noticeable. The extension of the hand is still possible, but the resistance of the extensor muscles is very easily overcome. The power of flexion is better retained. Flexion, extension, and pronation of the forearm are possible, but the range of motion is limited and the movements are easily resisted. The upper arms are but slightly affected: the patient can raise them up well and he can resist passive movements with considerable energy.

Electrical examination shows that in all the muscles of the hand faradic excitability is greatly reduced. Galvanic excitability is also much enfeebled and the reaction of degeneration is present in many of the muscles. There is manifest lessening of faradic response in the muscles of the forearm, which is most noticeable in those that are most atrophied. In general the electrical reactions are much more feeble on the right than on the left side. At least a partial reaction of degeneration is present in all the muscles that are noticeably affected by the disease.

Muscular atrophies are rarer in women, but they occur. To show you how a corresponding condition may develop in the female, I present this second case. She is a florist, fifty-two years of age, who about ten years ago began to feel the formications and tendencies to cramp which preceded the development of her disease. These were at first localized in the fingers of the left hand; a year afterwards they developed also in the right hand. It is interesting to notice that the development of her symptoms bears a very characteristic relation to her occupation, in which the thumb and index-finger, especially of the left hand, are called into play in the making of artificial flowers. In rolling the gummed bands of paper or other material around the metallic stems of the flowers, the left thumb and index are in almost constant use. It was this thumb and index-finger which were first attacked and their muscles first began to atrophy. Despite the gradual atrophy of the muscles she was able to follow her occupation for three or four years, but about six years ago she had to give it up definitely.

In the mean time the muscular atrophy spread beyond the hands. Two years after the commencemaent of the disease the forearm was affected and shortly afterwards the upper arm. When the patient came to the hospital some seven years ago, she was still able to accomplish certain feeble movements with her fingers. Despite careful electrical treatment for most of this time, the condition of her muscles has not been improved in the slightest. About seven years after the beginning of the disease her arms had become completely helpless and inert. As she says herself, they are as thin as a skeleton's. Fibrillary tremors are noticeable in the deltoid region and at the back part of the arm. These are due very probably to fibrillary contractions of the rudimentary remains of the atrophied muscles. Some years ago she began to experience pains in her neck, at first in front, then at the sides, especially in the region of the upper border of the trapezius muscles. Soon it became impossible for her to move her head backward and forward. For nearly two years now the head, too heavy for its atrophied muscular support, sinks forward on the breast and the chin is supported on the sternum.

Family and Personal History.—There is absolutely no sign of any nervous affection in her family, as far as can be traced. Her mother died at fifty-eight of cholera, and her father, who committed suicide, had always been in good health. She had none of the maladies of childhood and has never had any of the infectious diseases. She began to menstruate at fourteen and her menstrual life continued regularly up to the age of forty-three. She was married at twenty and has had three children, two of whom died in childhood from measles and broncho-pneumonia. She has a married daughter, who formerly enjoyed very good health. For the last five or six years, however,—as a consequence, she thinks, of pregnancy,—this daughter has suffered from feebleness of the hands, and has from time to time been obliged to interrupt her occupation, which is also that of florist. The daughter, however, who is now thirty years of age, presents no signs of muscular atrophy.

The present state of our patient is very characteristic of a class of cases in which various dystrophic muscular conditions develop more frequently than in normal individuals. She is very small, badly developed, and with outward bowing of the legs. Her tibias

are deformed from rhachitis. She tells us that she began to walk late in infancy. She presents no signs of a deformed pelvis. Her lower limbs are normal in appearance but for the tendency to bowing, and, though she occasionally has felt passing pains in them during the last few months, they do not seem as yet to be affected by the muscular atrophy. Her muscular power is not diminished. She takes walks and goes and comes where she wishes without fatigue. There is a certain awkward hesitancy in her gait, due perhaps to the enforced absence of the compensatory swing of the arms which by habit becomes so necessary for normal walking in many people.

The trunk muscles are absolutely unaffected and the movements of flexion and extension are accomplished without awkwardness or fatigue. The muscles of her neck, however, and those which rise from the upper dorsal vertebræ are very markedly atrophic, so that the vertebral spines jut out very noticeably. The position of the head, bent far forward on the thorax, renders the cervical vertebræ still more salient. On the scapula the supraspinous and infraspinous groups of muscles are almost entirely absent, especially on the right side. The deltoids have become rudimentary in size, and this renders abortive any attempt to raise the arms. It is in the region of the neck and shoulders that the atrophic condition is most marked.

In the arms there seems to be but little absence of normal muscles. Further examination, however, shows that the power of the arm muscles is but slight: function is almost completely lost. The contradiction between the appearance of the arms and the power they possess is most striking. Their rounded contour, however, proves to be due to an œdema which exists in the upper arms, the forearms, and the hands. Close examination shows that it is present also in the face. This œdema has existed, the patient thinks, only during the last four or five years. She will not say positively, however, that there were not some traces of it even before she began to notice the loss of power in her arms. Besides the existence of this œdema, the small stature of the patient would lead us to suspect some affection of the thyroid body, and examination shows that it is practically completely absent. This, of course, explains the presence of the œdema, and shows that we have to do with a case of muscular atrophy complicated by myxœdema. A

cursory glance does not show that this is a classic case of progressive muscular atrophy, yet such it proves to be. Careful palpation makes it easy to detect the absence of the muscular masses usual in the forearm.

The muscles of the face are absolutely untouched.

The study of the reflexes shows that the knee-jerks are normal, and there is no unusual irritability of the Achilles tendon. There is complete abolition of the tendon-reflexes at the wrist and elbow. Objective sensation is unimpaired. There is no trouble with the sphincters, no visceral lesion, and no difficulty in swallowing or breathing.

The most interesting thing about this case is the combination of progressive muscular atrophy with myxœdema in a subject presenting all the signs of having suffered from severe rhachitis in childhood. These combinations of diathetic and nervous diseases are not uncommon. They often exhibit complications that make extremely difficult the diagnosis of the exact pathological conditions present. We are of late years, however, learning not only that these combinations of constitutional and nervous diseases are not infrequent, but that there is in most of these cases an underlying basis of defect in the essential nature of the organism which is prone to interfere with many organic functions. The nervous system, as the most delicate, is sure to suffer, but other functions dependent on the nicely balanced mechanism of intricate chemical and physical processes are also likely to be affected.

The study of these two cases shows very clearly that there does exist simple progressive muscular atrophy of the Duchenne-Aran type and that it is an absolutely independent morbid entity. Professor Charcot has indicated its clinical and anatomical limits. The disease is absolutely distinct from amyotrophic lateral sclerosis. It is much more frequent than has generally been considered, so that it is of clinical importance even to the general practitioner.

As to the treatment of the affection very little can be said. As soon as the first symptom declares itself, all overwork of the muscles especially involved should be avoided. The two patients whom we have seen illustrate very well the specialized character of occupation which in many cases has been known to precede the manifestations of progressive muscular atrophy. Patients should at once be put into the best possible physical condition. General tonics,

good hygiene, plenty of fresh air, and a liberal diet are important measures. In the exhibition of tonic drugs, while strychnine may be of use, it should not be pushed to its physiological effect or it may do harm. This becomes clear from the fact that it is liable to increase the fibrillary tremors that are so marked a feature of the beginning of the disease. Mild applications of galvanic electricity or a not too rapidly interrupted faradic current have frequently been thought to do good. Hydrotherapy also is not without effect in retarding the progress of the disease. Something may be accomplished by prophylaxis. The disease is prone to occur in families, especially among brothers and sisters. Where one member of a family is known to be affected by the disease, other members of the family should be counselled not to engage in an occupation which requires persistent and delicate co-ordination of a number of muscles. It is not probable that this precaution will absolutely prevent the occurrence of progressive muscular atrophy, but it will at least put off its beginning and retard its course. It would seem that the disease is really due to a predisposition consequent on defect in the original germinal material given to the members of certain families. Persons in whose families cases of progressive muscular atrophy have occurred should be warned of the necessity of keeping themselves in as good general health as possible and of avoiding worry and nervous strain. They must be content to take life easy, and must avoid as far as possible the tension of concentrated effort and the wear and tear of the struggle for existence.

Surgery

GLUTEAL ANEURISM; CERVICAL LYMPHADENITIS; CARCINOMA OF THE RECTUM; ADENOMA OF THE MAMMARY GLAND; THYROIDECTOMY FOR CYSTIC GOITRE; NEURECTASY OF SCIATIC NERVE; CONCUSSION OF THE BRAIN; TRACHEOTOMY.[1]

ABSTRACTS FROM THE PUBLIC SATURDAY CLINICAL LECTURES DELIVERED AT THE GERMAN HOSPITAL.

BY JOHN B. DEAVER, M.D.,

Surgeon-in-Chief to the German Hospital, Philadelphia.

To obviate the necessity of constant repetition of aseptic details, the reports are prefaced by a general description of the technique usually employed. Variations due to accident or special conditions are mentioned under the reports of the cases in which they are necessary.

The constant aim in the clinic is to follow the principles of asepsis rather than those of antisepsis. The four principal sources of infection—the air, the hands of the operator and his assistants, the field of operation, and the instruments and dressings which come in contact with the wound—are all rendered as sterile as possible, prior to the operation, in the following manner:

The field of operation is first scrubbed with a stiff brush and soap and water. The part is shaved. Then it is again scrubbed with turpentine, soap, and water, and washed thoroughly with potassium permanganate, followed by a solution of oxalic acid, and this in turn by a bichloride of mercury and carbolic acid solution. The

[1] The material for these clinical lectures was secured from reports furnished by Drs. H. G. Godfrey, A. G. Ellis, and M. J. Cooley, Jr., in competition for a prize offered by Dr. Deaver for the best account of his clinics. The notes were edited, and the subsequent histories added, by Dr. A. D. Whitting, assistant surgeon to the German Hospital, while Dr. A. O. J. Kelly, pathologist to the hospital, inserted the pathological memoranda. The above reports form the first of a series of cases by Dr. Deaver which will appear in future issues of the CLINICS.

part is then covered with towels wet with the bichloride and carbolic acid solution. These are allowed to remain in place until the time of operation, when they are removed and the part rinsed with bichloride solution, alcohol, and sterile water. In all but emergency cases, where time will not allow, the patient is given a tub bath before this procedure is carried out.

After the patient is placed upon the operating-table, he is covered with warm sterilized blankets, the field of operation alone being left uncovered. The blankets are then covered with aseptic sheets and towels, to prevent the operator or his assistants coming in contact with septic materials, and also to afford an aseptic surface upon which instruments and materials may be placed within easy reach of the operator. The surgeon, assistant, and Sisters working in the operating-room bathe and don clean underwear immediately before going into the operating-room.

The hands of the operator and his assistants are prepared as follows. No jewelry is allowed to be worn. The finger-nails, if long, are pared. The aseptic suits are then put on, and the hands and forearms thoroughly scrubbed with a stiff brush, hot water, and green soap. After a careful scrubbing, the finger-nails are scraped with a blunt instrument and the hands again scrubbed. They are then washed in turpentine, the latter being removed by an antiseptic liquid soap and water. The operator and first assistant then cover their heads with a strip of sterile gauze, and wash their hands and forearms thoroughly in solutions of potassium permanganate, oxalic acid, alcohol, bichloride of mercury, and sterile water. The operator and all those who handle anything which comes in contact with the field of operation wear rubber gloves. During the operation the gloves are cleansed by frequent washings in sterile water.

The suits worn by the operator and his assistants consist of white bleached muslin trousers and sleeveless coats of the same material. They are sterilized by subjection to steam under fifteen pounds' pressure for twenty minutes. Each suit is covered by an aseptic gown before each operation. In all cases in which the operator works in the sitting posture he dons a rubber apron, over which the aseptic muslin apron is placed. The suits of the Sisters who act as assistants consist of long sterile aprons of muslin, asepticized in the same manner as the other suits. The gloves worn by the operator and his assistants are boiled for fifteen minutes in sterile

water. All towels used during the operation are subjected to steam for twenty minutes under fifteen pounds' pressure. The gauze used for dressings is treated in the same manner, but that used for cleansing the wound (thus taking the place of the sponge) is treated in the sterilizer for an hour and a half, and then again for twenty minutes before each operation. The cotton used in the dressings is placed in the sterilizer for twenty minutes under fifteen pounds' pressure.

All instruments except the knives are boiled in a one per cent. solution of sodium bicarbonate for fifteen minutes. The blades of the knives are wrapped in cotton and then placed in the boiling soda solution for a few minutes before the operation begins.

Silkworm gut is boiled in sterile water for twenty minutes and then placed in alcohol until needed. Catgut, plain and chromicized, is not used to any great extent. Silk is boiled in sterile water, the length of time being regulated by the size of the silk used.

All glass vessels, drainage tubes, etc., are boiled in sterile water. Other basins and vessels are thoroughly washed with soap and hot water, and then filled with bichloride solution until needed. They are then rinsed with sterile water and filled with the solutions required. The air of the operating-room is rendered as sterile as possible by means of formaldehyde gas, which is generated in the room at the close of each day's work. The floors are washed with bichloride and carbolic acid solutions, and frequently mopped during the operation. The operating-table is thoroughly scrubbed with bichloride and carbolic acid solutions, and rinsed after each operation.

The towels are replaced during the course of the operation, as they become soiled. The instruments and the hands are washed during the operation in sterile water contained in basins placed at the side of the operating table. This water is frequently changed by the Sister charged with that duty. The external wound is frequently washed with sterile water. In abdominal operations the pubes is covered by gauze moistened in a solution of bichloride of mercury and carbolic acid.

In dealing with septic foci, the pus is mopped away by gauze carried on long dressing forceps to avoid contamination of the hands of the operator and the external wound. The abscess cavity is sponged by dry sterile gauze and by iodoform gauze in the same

manner. The sterile towels are frequently changed; the instruments exposed to the infection are washed in sterile water. The hands of the operator are washed in a solution of bichloride of mercury and carbolic acid.

In abdominal operations *the wound* is rendered bloodless before the peritoneum is opened, and is washed from time to time during the course of the operation with sterile water. After suture of the wound it is washed with a solution of bichloride of mercury.

The patient is then raised, the wet coverings are removed, and the back is thoroughly dried with sterile towels. The wound margins are protected from septic discharges from drainage-tubes by a rubber dam, which is slipped over the neck of the tube and pinned by its four corners over a mass of iodoform gauze upon the mouth of the tube.

The same technique is observed in the redressing as in the operation itself. (Dressings of wounds about the mouth will be considered later under an appropriate case.)

The anæsthetic is administered upon a piece of sterile gauze folded in a narrow pad.

Four Sisters are on duty in the operating-room, of whom one prepares the instruments, ligatures, and dressings; the second accounts for the sterile gauze packing and sponges; the third replenishes the basins; and the fourth accounts for the discarded gauze.

The following is the usual method of closing the wound in all cases of abdominal section. Variations are mentioned under each case in which they are required. Through-and-through sutures of silkworm gut are first passed with a straight or Reverdin needle, the separate layers of tissue being picked up in advance of the needle so that all layers will be included in the stitch. The muscular structures are then coapted and the sheath of the rectus secured by means of a continuous silk or chromicized catgut stitch. The silkworm-gut sutures are then tied and the dressing applied. Where drainage is necessary, the wound above and below the drain is sutured with through-and-through silkworm-gut stitches, loose sutures being left at the site of the tube or gauze, which may be tied after the removal of the drain if it be thought advisable. This method generally results in union without the formation of much granulation tissue.

The dressing consists of a layer of gauze moistened in a solution

of bichloride and carbolic, covered by layers of dry gauze and absorbent cotton, all of which have been sterilized as described. The dressing is fastened in place by means of adhesive strips, and a many-tailed bandage is applied. This is also secured with adhesive strips. This dressing is used in all cases where applicable, the roller bandage being used instead of the many-tailed one after all operations except those upon the abdomen. In abdominal cases a sterilized towel is placed over and pinned to the bandage.

LIGATION OF THE RIGHT INTERNAL ILIAC AND RIGHT PROFUNDA FEMORIS ARTERIES FOR A GLUTEAL ANEURISM; EXCISION OF THE ANEURISM FOUR WEEKS LATER.

E. S., male, white, aged eighteen years. The aneurism occupies the right buttock and extends anteriorly over the perineum to the margin of the scrotum. It has progressively increased in size. It is of the variety described as cirsoid; while the sciatic, obturator, pudic, gluteal, and other vessels supply the mass, the gluteal is the principal afferent vessel, whence it is called a gluteal aneurism. The skin over the mass is thick, irregular, and slightly livid. At the inner end of the gluteo-femoral fold the sac is elongated, hanging pendulous between the thighs. On palpation pulsation is noted, and on auscultation there is marked bruit.

Ligation far away from the area of the disease is the present treatment. By ligating the internal iliac artery in the pelvis and the right profunda femoris in the thigh, anastomosis through the circumflex and perforating branches will be cut off. Local interference is contraindicated by the extensive dissection necessary and the danger of uncontrollable hemorrhage. After the primary operation, a plan for the excision of the mass of pathological vessels to prevent recurrence by anastomosis will be considered.

Operation.—Ether anæsthesia, followed by oxygen. The patient was in the Trendelenburg position. Incision was made within the outer border of the right rectus muscle, in the lower half of the space between the umbilicus and the pubes. Skin, fasciæ, and the sheath of the rectus muscle were incised, the wound was sponged, and the vessels caught. The deep epigastric vessels were exposed within the rectus muscle, ligated above and below, and divided between the ligatures. The fascia transversalis and peritoneum were lifted by forceps and divided. Coils of bowel were excluded

from the field of operation by moist gauze. The edges of the wound were separated by retractors. The peritoneum over the internal iliac artery was divided carefully, to avoid the ureter, and the sheath of the vessel was opened. A double ligature of silk was thrown around the artery by an aneurism needle, care being taken to avoid the vein and ureter. The vessel was tied about one inch from the pelvic brim, and the sheath of the vessel and the peritoneum were closed by a continuous silk suture. The gauze packing was removed, the omentum spread over the intestines, and the patient placed in the horizontal position. The wound was closed without drainage.

An incision about three inches in length was made from a point below Poupart's ligament, external to the vessels, obliquely downward over the femoral vessels. The skin and fasciæ were divided and the vessels caught. The profunda was located branching low down from the common femoral. The sheath of the vessel was opened, a ligature passed by an aneurism needle, and the vessel ligated. The wound was closed by interrupted sutures of silkworm gut. Dressings of gauze moistened with a solution of mercuric chloride (one to two thousand), dry gauze, and gauze-cotton pads were applied, being fixed by adhesive strips and by a bandage, which was bound by adhesive strips.

Examination following the operation showed a complete disappearance of pulsation and bruit.

In three weeks the pulsation and bruit, which were plainly observed prior to the operation, have disappeared, due to ligation of the vessels. Only slight contraction in the volume of the mass has taken place. It is yet pendulous and redundant. Greater contraction might occur after a while; but sufficient time has elapsed to indicate that the tendency towards contraction is but slight, and we are justified in seeking means to expediate cure, as the mass is large and redundant and there is danger of recurrence by anastomosis. Inquiry into the literature bearing on gluteal aneurism, which dates from pre-aseptic days, shows a high mortality. No case of excision of a gluteal aneurism is reported.

An attempt to secure coagulation by the injection of a two per cent. solution of gelatin in normal saline solution has lately been made by Dr. W. W. Keen. This was in the case of an aneu-

rism of the abdominal aorta, which had ruptured. A huge mass was found occupying the left side of the abdominal cavity, which exploratory cœliotomy showed to be a retro-peritoneal clot. The wound was closed without disturbing the clot, and the gelatin and saline solution were injected into the subcutaneous tissues of the chest.

The mass could not have been extirpated primarily, and as a secondary operation extirpation should be done before there is any likelihood of a recurrence through collateral anastomosis. Hemorrhage may be primary, consecutive, secondary, or recurrent, the last named being due to the establishment of collateral circulation. The secondary operation, to be successful, must anticipate this extensive anastomosis.

The treatment is comparable to excision of a nævus or to the radical cure of a circumscribed aneurism in the extremities,—viz., cutting off the blood-supply by proximal and distal ligation, and followed by excision of the aneurismal sac. The blood-supply must first be checked at a distance from the growth.

The dressings were removed from the abdominal and femoral wounds, which showed primary healing.

Operation.—Ether anæsthesia, followed by oxygen inhalations. The patient was lying on the left side, the thighs flexed on the abdomen, the legs flexed on the thighs and supported upon a stool. An incision was made from below upward along the inner border of the aneurism, at a distance of about one and one-half inches from the groove between the nates. In order to occupy less time, a V-shaped mass was to be removed. The primary incision extended along what was, as the patient lay on his side, the most dependent border of the tumor. Dissection was proceeded with in a direction forward and outward. As the depths of the mass were invaded, considerable vascularity was encountered, and the hemorrhage became very free, but was controlled by the application of forceps and by pressure with hot gauze. The primary incision was tightly packed with hot gauze; the forceps remained *in situ*. A second incision was then made upward and outward along the gluteal fold, beneath the pendulous tip of the tumor. Dissection was continued inward and forward towards the primary incision. The wedge-shaped mass was removed by cutting between the upper ends of the incisions. The wound appeared covered with hæmo-

stats. The vessels were immediately tied securely with heavy catgut. Fifty-four ligatures were applied. The cavity of the wound was tightly packed with large pieces of iodoform gauze, held in place by several stitches of silkworm gut passed through the edges of the wound. The gauze will not be removed until granulation is well under way. The abdominal and femoral regions were well covered with gauze and cotton dressings. The upper thigh, lower abdomen, and right buttock were included by a roller bandage, which was firmly secured by adhesive plaster, especially in the vicinity of the anus. The operation occupied forty-five minutes. To avoid shock the large volume of blood was restored by the intravenous injection of two quarts of normal saline solution.

The vascularity of the growth showed the danger attending a primary incision. It should serve as a practical warning against the rash excision of nævi, for which ligation can often be done with advantage.

The patient made an uneventful recovery.

CERVICAL LYMPHADENITIS.

D. H., male, white, aged twenty-seven. A well-marked swelling occupies the right submaxillary region and extends behind the ear. On palpation there is found extensive adenitis of the superficial and deep lymphatic chains in the upper half of the neck, with slight involvement below.

Operation.—Ether anæsthesia, followed by oxygen. The patient was in the horizontal position, with shoulders raised by a low pillow and the head rotated to the left. An incision was made over the external carotid artery, extending from a point anterior to the lobule of the ear to the level of the thyroid cartilages and slightly curved inward; it was carried through the superficial fascia, dividing the fascia and platysma myoides. The external jugular vein was caught by forceps, tied above and below, and divided between the ligatures. The superficial glands overlying the sternocleidomastoid muscle were removed. The deep fascia was opened and dissected from this muscle, and the muscle and fascia were retracted outward. The glands of the deep chain were removed from the submaxillary and the superior carotid triangles. The lingual, facial, and internal jugular veins, the external carotid

artery, the hypoglossal, the descendens hypoglossi, and the spinal accessory nerves, and the digastric muscle were exposed and demonstrated in the course of dissection. The vessels occluded by the hæmostatic forceps were tied and the wound was sponged dry. A rubber drainage-tube was secured in the upper and lower angles of the wound, which was closed by interrupted sutures of silkworm gut. The tube was then washed out by a piston-syringe. Dressings of dry gauze and absorbent cotton were applied and fixed by a gauze bandage.

The patient was discharged on the nineteenth day, with a small sinus at the site of the drainage-tube.

In operations on the neck the sternocleidomastoid muscle is the guide to the vessels. In any region in which damage may be done by indiscriminate dissection, expose the landmarks by a free incision. Keep the tissues dry by frequent sponging, that all the structures may remain visible. In excising glands guard the surrounding structures by keeping the scalpel close to the body to be removed.

Drainage is always advisable, since in this region obliteration of the dead spaces cannot be accomplished by pressure, and some reactionary hemorrhage is frequent. The tube may be removed under complete aseptic precautions in twenty-four hours.

In several cases of amputation of the breast in which a stump of the lesser pectoral muscle remained, free drainage from the upper inner quadrant of the dissection was interfered with, and as a result a hæmatoma formed there. The blood-clot may be absorbed, but it is liable to become infected. Hence it is good practice to remove the pectoralis minor muscle completely or to carry a long drainage-tube well beneath the clavicle.

Cervical lymphadenitis is in the majority of cases tubercular. Many cases arising in the debilitated are non-tubercular. Extensive adenitis following influenza has of late been frequently seen.

Methods of treatment by internal and external medication may be used. More favorable results follow well-chosen internal treatment. Effective alteratives are syrupus ferri iodidi in full doses (from half a drachm to one drachm) and cod-liver oil in large doses. If gastric irritation result from its use, the following may

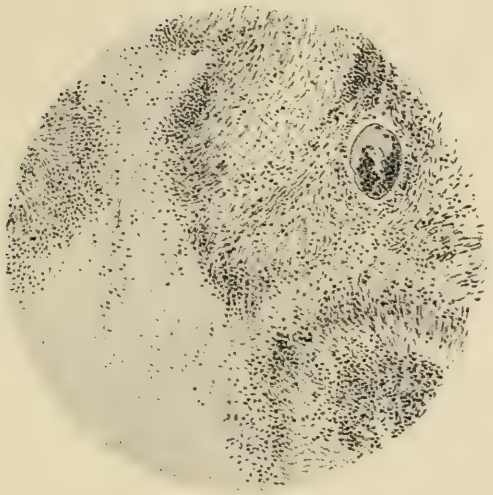

FIG. 1.—Tuberculous adenitis of the neck. The section shows the periphery of one of the larger foci of necrosis, about which there are numerous epithelioid cells and round cells. A giant cell is also seen. Oc. 3, Ob. C.

FIG. 2.—Adenocarcinoma of the rectum. The section reveals irregular and atypical proliferation of the mucous glands of the rectum. The epithelial lining in many places consists of several layers, while in other places the basement membrane has been ruptured, and irregular proliferation of the epithelial cells has occurred in the muscular layer of the bowel, where there were a number of dense carcinomatous nests. Oc. 4, Ob. numerical aperture, 0.65, Zeiss.

be given in capsules thrice daily: ferri iodidi, gr. 1½; acid. arsenosi, gr. $^1/_{40}$; hydrarg. bichlor., gr. $^1/_{40}$. Abundant and nutritious diet and systematic out-of-door exercise are necessary. Judicious stimulation by alcohol may prove beneficial. In a case seen with Dr. Pepper, a child with extensive cervical adenitis, most remarkable results followed the use of thyroid extract.

External treatment by inunction and electrical stimulation is not regarded favorably. Very considerable irritation and traumatism may result.

These constitute a most difficult class of cases for surgical treatment. Enucleation is not always advisable. In early local tubercular adenitis operation is indicated. In non-tubercular cases treatment should be conservative. Deformities and pressure-effects may indicate operation.

On examination the specimen was found to consist of two masses, measuring respectively five by three by two and one-half centimetres and five by three by two centimetres. They are of solid composition, but somewhat soft; of smooth surface, and reddish color. On section they are reddish to whitish, opaque, and in places somewhat softened. Microscopic examination revealed the well-known lesions of tuberculous adenitis,—numerous epithelioid cells, giant cells, round-cell infiltration and foci of necrosis. (Fig. 1.)

MODIFIED KRASKE OPERATION FOR EXCISION OF AN ANNULAR CARCINOMA OF THE RECTUM.

B. A., male, white.—Operation was done ten days ago.

Two similar cases have been treated here recently, and in both defecation was as normal after as before the operation. In each case the continuity of the bowel was restored and in neither was a preliminary colostomy performed.

There is no worse morbid condition than carcinoma of the rectum. The patient's suffering is unbearable. The constant desire to defecate and the inability so to do, the intense anorexia, and the terminal profound cachexia present a frightful picture.

As to the operative methods for its relief surgeons differ. Some advise a radical operation at one step; others suggest a preliminary colostomy. The radical operation is applicable only to the early cases, where the removal of the growth may result in complete and most satisfactory cure.

Early diagnosis of the situation and extent of the growth is the first essential. Examination by the finger within the rectum may be sufficient. We would have no hesitation in opening the abdomen for this purpose and, if the growth were too high to remove by incision over the sacrum, we would not hesitate to remove it through the abdomen. Those cases in which inguinal or lumbar colostomy must be done are regarded as too extensive for radical operation.

Inguinal colostomy should not be performed if at all avoidable. Its disadvantages are set forth in the last volume of S. D. Gross's "Surgery" in a most vivid description, in the course of which the author states that it should never be done. The operation as now performed has yielded good results in the experience of some surgeons. Dr. W. W. Keen, who has done much in this line of work, recommends a preliminary colostomy in all cases.

The important consideration is early radical operation. The same principles of treatment as in carcinoma of the breast apply likewise to the operation for malignant disease of the rectum. In a case of carcinoma of the clitoris the glands of both groins were removed before attacking the primary disease. The condition of the patient improved marvellously after operation, and a few days ago she returned home.

This is the second redressing of the wound. A sterile Kelly pad was placed beneath the patient. The field was surrounded with sterile towels and the dressings were removed. The wound was washed with sterile water and the iodoform gauze was removed from the wound. A thick rubber drainage-tube was seen protruding through the anus. Stitches were removed from the upper and lower angles of the incision. The open wound was in the median line, about three inches in length, extending downward from a point about two and one-half inches below the level of the posterior superior iliac spines. The rectum, covered with granulations, was seen in the depths of the wound. The wound was repacked with iodoform gauze, which was carried into the wound by long dressing-forceps. Dry gauze dressings were applied and fixed by a roller bandage, which was bound by adhesive strips.

Method of Operation.— The incision is made in the median line posteriorly down to the anal margin. The coccyx is removed. The sacrum is uncovered and the lower two or three pieces of this bone are disarticulated by an osteotome and pried from their

position; the ligamentous attachments are cut away with scissors. The segments of the sacrum are removed carefully, to avoid injury to the third sacral nerve, which supplies the anal sphincter. The anterior muscular attachments are divided after the bone is loosened, to avoid much hemorrhage. The bowel is exposed along with the levator ani muscle, and is incised as far as possible from the area of the disease above and below. The peritoneal cavity may be opened, the mesosigmoid cut, and the bowel brought down. So much of the sigmoid may thus be excised that it is converted into a straight tube. The carcinoma having been excised, the peritoneum may be closed by suturing it to the serosa of the bowel. The gut is closed by double rows of silk sutures, the first including the muscular and submucous layers, the second being a Lembert stitch of the peritoneal coat. (Fig. 2.)

All patients thus treated have recovered the function of the rectum completely. The last case had a persistent fistula which communicated with the rectum. Defecation by the normal passage was not possible until the sphincter ani was dilated. The sinus then closed.

In this case the sphincter was stretched. A tube was inserted to relieve tension at the line of suture.

All our cases thus treated have recovered; one after three years shows no sign of recurrence.

ADENOMA OF THE RIGHT MAMMARY GLAND.

Miss B., white, aged twenty-three years.—On palpation a mass the size of a pigeon's egg is found in the lower half of the right breast. It is freely mobile on the underlying structures and is not adherent to the skin.

In the diagnosis of growths of the breast it should be remembered that ninety per cent. are malignant. This should be considered with the age of the patient and the physical characteristics of the growth. Carcinoma may occur in the young. Tubulated carcinoma of the breast in a young girl and carcinoma of the rectum in a male twenty-three years of age were operated on here not long since.

The radical operation is especially applicable to the early cases; hence in this case, although the growth has the clinical character-

istics of a simple adenoma, it will be removed for microscopic diagnosis.

Operation.—Ether anæsthesia, followed by oxygen. The patient was in the horizontal position. Incision was made in the course of the lactiferous ducts directly over the growth, which was dissected free and removed. The wound was closed by interrupted sutures of silkworm gut, several strands being introduced between the stitches for drainage. Dry dressings of gauze and cotton were applied and fixed by a roller bandage. The stitches were removed on the tenth day, and the patient was discharged.

The *mammary gland* is situated between two layers of the superficial fascia, of which the upper gives off fibrous septa which pass through the gland and support the glandular structure. These are known as the "suspensory ligaments of Sir Astley Cooper." Contracture of these fibrous septa and of the lactiferous ducts will produce retraction of the nipple.

The lymphatic system is made up of a superficial and a deep set of vessels. The superficial channels form a rich plexus in the submammary connective tissue. This is often the site of a submammary cyst. The deep lymphatics are walled off by the pectoral fascia. Both systems drain into the axilla. Extension of a malignant growth to the pleura or the mediastinum may take place through lymphatic vessels perforating the chest-wall. If a malignant growth is situated in the upper outer quadrant of the breast, axillary involvement is rapid; if in the lower inner quadrant, extension to the mediastinum is more frequent. Hence the condition must be met by early diagnosis and radical operation. Little disability follows the removal of the muscular structure if the clavicular fibres of the great pectoral muscle be preserved.

Examination of the excised growth revealed it to be a tumor three centimetres in diameter, firm to the touch, whitish in color, and surrounded by a small amount of adipose tissue. On section it was grayish-white and somewhat opaque. On microscopic examination it was found to be a mixed tubular and alveolar adenoma. Some of the alveoli were the seat of cystic dilatation, whereas others showed proliferation of the epithelial cells into their interior. This epithelial proliferation indicates the wisdom of early operation in this as in all apparently benign tumors of the breast, and it suggests also the propriety of further observation of this patient. (Fig. 3.)

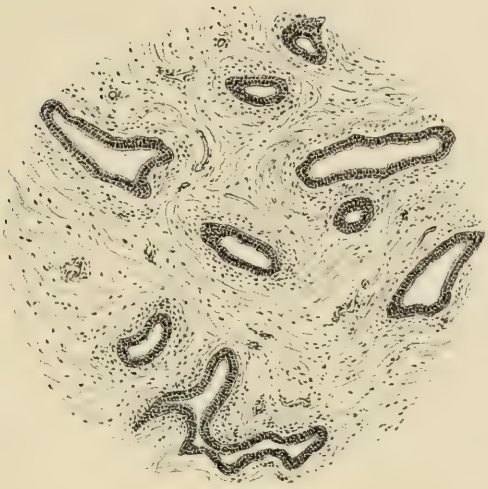

FIG. 3.—Adenoma of the breast. The section reveals a great overgrowth of connective tissue and a marked proliferation of the acini and ducts. Histologically the latter are typical, but in some sections they were much compressed, whereas in others they were the seat of slight cystic dilatation. Oc. 3, Ob. AA.

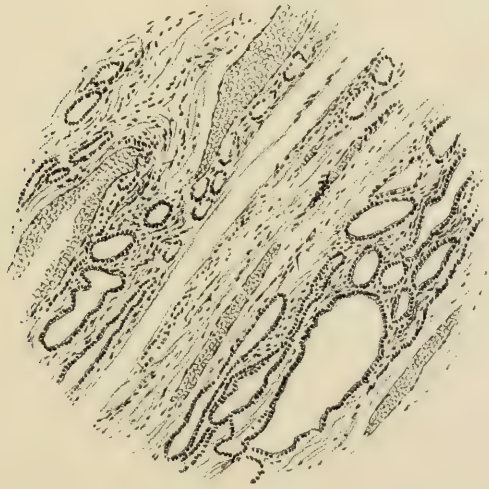

FIG. 4.—Cystic goiter. The section is from the periphery of one of the large lobules, and reveals, within a rather dense layer of connective tissue, a number of acini of varying size, lined with a single layer of epithelium and containing colloid material. Oc. 4, Ob. numerical aperture, 0.65, Zeiss.

PARTIAL THYROIDECTOMY FOR CYSTIC GOITRE.

W. S., female, aged twenty-three years.—A bilateral swelling, slightly more prominent to the left side, of some years' duration, occupies the lower and median region of the neck. The mass rises during inspiration when the patient is in the recumbent position. This is a simple goitre, a local condition only, in contradistinction to exophthalmic goitre, in which the enlargement of the thyroid is associated with characteristic constitutional symptoms.

Surgical treatment will give satisfactory results in selected cases, but it is not always applicable. Enlargements of the thyroid in young girls may disappear with the the onset of menstruation. Pressure-symptoms and rapid increase in size of the goitre, as were present in this case, are indications for surgical interference. A portion of the gland must always be allowed to remain, to avoid post-operative myxœdema. Vascular goitre may be mistaken for an aneurism.

Our experience with thyroidectomies has included fifty cases. Kocher, of Berne, in July, 1896, had performed one thousand and six operations, and since that time has done hundreds more. Dr. Rodman tells us that it is Kocher's practice to make a Y-shaped incision, a straight median incision with a branch on each side extending out along the inner margin of the sternocleidomastoid muscle. A semilunar incision is used in large growths only. All such operations are performed under cocaine anæsthesia.

In this case a semilunar incision with its convexity downward will be made. This will leave as little scar exposed as possible.

Operation.—Ether anæsthesia, followed by oxygen. The patient was in the horizontal position, the shoulders being raised by a pillow and the head falling backward to expose widely the structures of the neck. A semilunar incision was made from the left to the right, the convexity reaching to the lower border of the tumor. Skin and superficial fascia were incised and dissected upward. The anterior jugular veins were exposed and tied. The deep fascia overlying the sternocleidomastoid muscle was dissected free by the scalpel and the finger, and the sternohyoid and sternothyroid muscles were exposed. These muscles were divided longitudinally, and they were retracted on each side. The goitre, enclosed in the pretracheal layer of the deep fascia, was exposed; the right recurrent

laryngeal nerve was sought in the tracheo-œsophageal groove and guarded. The inferior thyroid vessels and the middle thyroid veins were caught in forceps and divided. The capsule was separated and the growth was readily enucleated, the recurrent laryngeal nerve of the right side being guarded by gauze. The capsule and the cortex were incised above the middle of the mass. The medulla of the gland was transfixed by a pedicle-needle carrying a double silk ligature. The ligatures were tied and their ends were joined again, a pedicle stump being thus formed. The mass was cut away below the pedicle and the pedicle stump was trimmed with scissors. The goitre was cystic on each side. The vessels included in the forceps were tied off with catgut. The wound was washed with sterile water, and the bleeding points in the pedicle stump were caught and tied off with silk. The wound was again washed with hot sterile water, sponged dry, and carefully inspected for bleeding. The wound was closed, without drainage, by interrupted sutures of silkworm gut. Dressings of gauze and cotton were applied and fixed by a roller bandage, which included the neck and the front of the chest and passed beneath the axillæ. (Fig. 4.)

The stitches were removed on the tenth day, and the patient was discharged on the twelfth.

Dr. Rodman, at the request of Dr. Deaver, spoke of a visit to Kocher at Berne during July, 1896.

Kocher had at that time performed one thousand and six thyroidectomies. Upon request of Dr. Rodman, an appointment was made for the next morning at six o'clock, when six such operations were to be done. These were completed within two hours, as a result of the excellent co-operation of the trained assistants, to whom Kocher passed each case successively as soon as enucleation was complete. Local anæsthesia was used in every instance. On transfixing and tying off the pedicle it was the custom to request the patient to articulate, in order to be assured that the recurrent laryngeal nerve was not included in the ligature. Dr. Rodman did not regard the local anæsthesia favorably. Considerable hemorrhage attended the operations, despite the skill of the assistants. Fifty or seventy-five silk ligatures were used in each case, and in one of these the bleeding was sudden and most profuse. A visit to the wards showed that this extensive use of silk was not attended with ill results.

As to its etiology, Kocher was of the opinion that the drinking-water was a factor; to this he attributed its endemic distribution in the Alps and Himalayas.

Of goitre in young girls, Dr. Rodman had two cases in which the tumor disappeared with the establishment of menstruation. Both were locally treated with biniodide of mercury ointment, applied after the method in vogue in India, by inunction while the patient was exposed to the direct morning sunlight.

NEURECTASY OF THE RIGHT SCIATIC NERVE.

E. S., male, aged thirty-nine years.—Patient was transferred from the medical wards, where his ailment had not responded to treatment. On inspection, the scars of the cautery over the course of the right sciatic, the internal popliteal, and the posterior tibial nerves are seen, extending from the gluteofemoral fold to the calf of the leg.

The great sciatic nerve may be reached by an incision through the middle of the thigh posteriorly between the hamstring muscles; or by a higher incision at the gluteofemoral fold, between the insertion of the gluteus maximus muscle and the biceps. It takes the name of sciatic at the lower border of the great sacro-sciatic foramen beneath the pyriformis muscle. It may pierce this muscle or may bifurcate high up. Ordinarily it divides into the external and the internal popliteal branches in the middle or lower third of the thigh. The line of the nerve can be marked out from a point midway between the tuberosity of the ischium and the great trochanter, to the middle of the popliteal space.

Operation.—Ether anæsthesia, followed by oxygen inhalations. The patient was supine in the horizontal position, the right thigh being slightly abducted. The gluteofemoral fold was located. An incision two inches in length was made downward in the course of the nerve from the gluteal fold. The tissues were deeply incised; then by the finger within the wound the nerve was grasped and brought to the surface. Traction was made by the finger covered with gauze, in a direction both upward and downward. The extremity was partly lifted by traction on the nerve, and while maintaining traction the leg was forcibly extended. This was continued for about five minutes. The nerve was then returned within the wound, which was lightly closed, without drainage, by

interrupted sutures of silkworm gut. The wound was dressed with dry gauze, which was bound by a roller bandage fixed by adhesive strips. The patient was returned to the medical wards, no relief having been afforded.

CONCUSSION OF THE BRAIN.

A. M., male, white.—This patient had just been brought into the house, and had been sent into the clinic with the diagnosis of a fracture of the base of the skull. Following injury there had been a period of unconsciousness, and hemorrhage from the nose and the ear. In the clinic the patient began to show signs of returning consciousness. He had not been seen before; but from the general appearances the case was considered rather one of concussion of the brain, with evidences of resultant cerebral irritation.

Fractures of the base of the skull involve most frequently the middle and posterior fossæ. The petrous portion of the temporal bone is also commonly involved. Hence hemorrhage from the ear is a frequent symptom. The ear consists of an external, a middle (containing the chain of ossicles joining the membrana tympani, and communicating with the mastoid cells and the nasopharynx), and an internal portion, the osseous and the membranous labyrinths, which are separated by a liquid called the perilymph. Hence hemorrhage from the external ear does not occur unless the external tympanum is ruptured. This hemorrhage is followed by an escape of cerebro-spinal fluid. Search should be made for symptoms due to involvement of the facial and the other cerebral nerves at the base of the brain. Hemorrhage alone cannot be regarded as a sign of fracture at the base of the skull in the absence of cerebro-spinal fluid, stertorous breathing, inequality of the pupils, facial palsy, and impairment of function of the anal sphincters.

TRACHEOTOMY.

W. A., male, white, aged fifty-seven years.—This patient was admitted two weeks ago, with a suicidal wound which entered the larynx through the thyro-hyoid membrane. The wound was closed by superficial sutures, a few deep sutures only being buried. We do not advise the use of buried sutures in the cartilaginous structures of this region; they invariably cut out. There are now two fistulæ, which permit the passage of air. These fistulæ will not

heal unless the larynx is put at rest. We will therefore freshen the edges of these fistulæ, suture them, and then do a temporary low tracheotomy. There is slight inspiratory stridor suggestive of the laryngeal diphtheria for which in times past tracheotomy was performed so frequently. There is also dyspnœa, shown by the depression in the supra-sternal notch. These symptoms indicate the presence of laryngeal obstruction.

Operation.—Ether anæsthesia, followed by oxygen inhalations. The patient was in the horizontal position with a Kelly pad beneath his shoulders. By an elliptical cut with scissors the margins of each sinus were freshened. The base of the track was explored, and was found to communicate with the larynx. The wounds were closed with continuous sutures of fine black silk. The head of the patient was depressed, and a small pillow was placed between the shoulders, the head being stretched over the pillow, thus allowing complete exposure of the lower neck. An incision two inches in length was made from below upward in the median line, reaching to the level of the cricoid cartilage. The skin and superficial fascia were cut. The deep fascia was divided between the anterior jugular veins, the transverse communicating branch being caught with forceps and divided, and a separation of the sterno-hyoid muscles forced. The thyroid body was lifted; the pretracheal fascia was incised, and the trachea was grasped and opened by dividing two or three rings with the point of the knife. There was at once a violent inspiratory rush of air, followed by spasmodic expiratory efforts. The tube was quickly introduced and secured with tapes. The end of the tube was covered with moist gauze to receive the ejected blood and mucus. The vessels were tied with catgut. The lower end of the wound was stitched. Dressings of gauze and cotton were applied, and fixed by a roller bandage including the neck and the axillæ. The end of the tracheotomy tube was loosely covered with moist gauze to act as a filter. The temperature of the patient's room should be 75° F. The patient developed a catarrhal pneumonia, and died on the tenth day.

The *position of the head* has much to do with the rapid and successful performance of the operation. The shoulders may be raised by placing a pillow or by a folded blanket under them. The head might be allowed to fall over the edge of the table, being maintained in this position by light support.

Tracheotomy for membranous laryngitis is now seldom done. It cannot be performed so rapidly as in this case. The edge of the tracheal wound should be retracted, and the false membrane should be dislodged by means of a hook or feather, or by forceps, and removed; otherwise, the membrane might be pushed down below the tube and occlude its lower orifice.

Intubation has largely taken the place of tracheotomy. Tracheotomy is the more certain operation for the relief of the urgent symptoms, yet intubation, in most cases, will suffice. It requires no anæsthesia and no cutting, is very quickly done, and is free from septic bronchitis and pneumonia, which complications make the mortality for tracheotomy operations so high.

A *high or low tracheotomy* may be done. In the high operation the trachea is opened above the middle lobe of the thyroid gland; in the low operation, below it. In children the high operation is advisable. Important anomalous vessels may encroach on the field of operation. The left brachio-cephalic vein, or the right common carotid, or the thyroidea ima artery may cross high in front of the trachea. These anomalous conditions are rare, yet the possibility of their occurrence should be borne in mind in order that accidents may not happen.

The method of the operation is most important. All incisions should be made from below upward. Skin and fascia are incised. The anterior jugular veins are first met. These veins are joined by a transverse branch below, just before they penetrate the deep fascia in the supra-sternal notch to join the internal jugular vein. It is well to remember that none of the important vessels lies anterior to the deep fascia. The surgical anatomy of this deep fascia is most instructive. A superficial layer invests the pretracheal muscles, extends outward enveloping the sternocleidomastoid muscle, and blends with the fascia roofing in the posterior triangle of the neck. A deep pretracheal layer forms a capsule for the thyroid body, envelops the trachea anteriorly, sheaths the carotid vessels, binds down the omo-hyoid muscle, and finally blends with the more superficial layers over the anterior scalene muscles. After the skin, the deep fascia is next incised, the pretracheal muscles are separated, and the pretracheal fascia is opened. The stridor caused by the inrush of air into the trachea is exactly simulated by the sound of the sudden emphysema that occurs in the pre-

tracheal tissues when this layer of fascia is incised. Many a young physician has been deceived by this sound into believing that he has opened the trachea, and has fixed a tube in the pre-tracheal connective tissue. While the trachea is moving up and down with rapid respiratory action, it should be grasped with a tenaculum and one or two rings should be opened by the point of the knife from below upward. The trachea should be steadied with blunt hooks and search made for false membrane. The tube should be guarded by a plunger, in order that its rough edges may not catch the walls of the trachea. Tapes should be placed upon the tube before its introduction.

If the patient has not been accustomed to out-door life, the sudden rush of cold air into the trachea might set up inflammation. Such a patient should be placed in a room with a high temperature and a moist atmosphere. The temperature of this patient's room will be 75° F. Moist gauze will be placed over the tube as a filter.

Pathology

THE RÔLE OF THE BLASTOMYCETES OR FERMENTS IN THE ETIOLOGY OF CANCER.

A LESSON AT THE CLINICAL INSTITUTE FOR SURGERY OF THE ROYAL UNIVERSITY OF ROME, DIRECTED BY PROFESSOR FRANCESCO DURANTE.

BY PROFESSOR DEMETRIUS RONCALI,

Assistant Professor of Surgical Pathology at the University of Rome.

GENTLEMEN,—The most interesting question in surgical pathology at the present day is the etiology of cancer. Our treatment of malignant conditions has in recent years progressed with giant strides, and in many cases cancer is no longer the hopeless affection that it was. As our methods of diagnosis have improved, the number of cases recognized as cancer has increased. This increase has been so marked in most parts of the world that there is in many countries serious question, in the minds of physicians and surgeons who see large numbers of cases of cancer, whether its more frequent occurrence is only apparent because of better diagnostic methods or the disease is actually much more prevalent than formerly. If we knew the cause of cancer, we should probably be able at least to limit its occurrence, and perhaps also be able to diagnose the condition with certainty earlier than at present.

Up to the present time two theories as to the origin of cancer have prevailed among medical men,—the embryonal and the parasitic. These two theories of etiology are not, as many people seem to think, absolutely exclusive the one of the other. It is not impossible that there may be a predisposing and an actuating cause for cancer. Embryonal inclusions may prove to be the parts of the animal organism most liable to take on luxuriant overgrowth as the result of parasitic irritation.

We owe the embryonal theory to Professor Durante, the head of our clinic. Struck by the frequency with which malignant tumors develop on the sites of mothers' marks, nævi, etc., Durante in 1874 came to a conclusion which attracted wide-spread attention. It was supported to a great extent by what was at the time known of histology and embryology. He declared that those parts of the human body which have preserved their anatomical embryonal characters up to adult life, or which have reacquired an embryonal character by some deviation of normal chemico-physiological activity, are the tissues from which spring all new growths, and especially the malignant new growths. Embryonal tissue elements remain included in the midst of tissues which are themselves well developed without giving the slightest hint of their existence. Some unnoticed impetus, it may be only a slight irritative stimulus, suffices to excite in these tissues a nisus generativus,—that is, a proliferative tendency, and neoplastic formation results. Professor Durante compares this awakening of cellular activity to that which heat excites in the cellular elements of the germinative spot of birds' eggs: as is well known, the cells of the egg remain absolutely inert from the moment they drop from the cloaca until the heat stimulus comes. A very little reflection leads to the conclusion that, however complicated may be the arrangement of the tissues which constitute the substance of an epithelioma, the genesis of cancer in any part of the organism is due to the reproduction of an embryonal condition in the cells which go to make up the neoplasm.

This interesting and suggestive theory ought not to be, in fact cannot be, excluded from our modern pathology. It is perhaps necessary to restrict somewhat its significance and to limit the tumors to which it applies to certain special varieties,—for example, the dermoid cysts, benignant organoid tumors, and tumors derived from tissues which are not normally present in the organs in which they appear. Durante's statement in his treatise on surgical pathology remains to the present day a great truth and contains a great principle. No neoplasm shows better than teratomata that embryonal remains represent the essential etiological element in the formation of neoplasms.

The best-known type of benignant organized tumors is the adenoma. This tumor can scarcely be considered to be of parasitic

origin, not because it is glandular, but because malignancy is wanting. It is difficult to understand how neoplasms can be malignant without some extraneous agent,—that is, a parasite. To admit that the same embryonal germ activity which, by causing proliferation during intra-uterine life, brought about the formation of our organic system may, while performing a similar function in the organism after birth, give rise to a product which is incompatible with life itself, is as much as to concede that physiological tissues may become pathological without essential change and that normal growth is a series of malignant neoplastic processes.

When an adenoma becomes malignant, or when it is transformed into an epithelioma, or when embryonic connective tissue becomes sarcomatous, we cannot help but think that, besides the perverted embryonic forces, parasites are at work in the tissues. In a word, the parasitic germ adds its force to the hitherto latent energy of the misplaced embryonal germ. The location of the parasite in an adenoma is due to the fact that this sort of tissue presents a *locus minoris resistentiæ*, which makes infection by the parasite and its rapid multiplication possible, and also allows the unbalanced epithelial elements in the part to take on such a rapid proliferative process. It is the parasitic irritation that adds the element of malignancy to the more or less physiological overgrowth of tissue that has taken place. If the leucocytes which at once find their way in large numbers into the irritated adenoma should be aroused to the cellular activity that would make of them fully-formed connective tissue, and if the fixed cells of the connective tissue of the part, together with the endothelium of the blood-vessels in the neighborhood, become affected by the same parasitic irritation, a sarcoma may develop in the midst of the adenoma. Something of the same kind occurs in teratomata when, as happens occasionally, they become metamorphosed into epitheliomata.

It is more than a quarter of a century since the parasitic origin of cancer was first suggested. In 1875 Nepveu pointed out that malignant neoplasms present all the characteristics of a parasitic infection. Since that period a great many different forms of parasites have been described as the cause of cancer. No one of them attracted wide-spread attention, however, until 1895, when the subject entered on a new phase. The history of parasitism in cancer is interesting not only because of the great practical im-

portance of the subject, but also because the progress herein made affords a striking illustration of the usual method of procedure in medical investigation. This history divides itself naturally into four periods,—first, that in which the observations made and the interpretations thereof were erroneous; second, the period of exact observation but erroneous interpretation; third, that of exact observation and interpretation, but in which the conclusions were not demonstrated experimentally; and, fourth, the period of exact observation and interpretation, with experimental demonstration.

The first period may well be called that of the bacterial theory of the origin of cancer. Nepveu described certain microbes which he found in epithelial tumors, though he did not ascribe to them any specific etiological action. Some ten or twelve years later, Rappin and others found still further forms of bacteria. Rappin isolated from the fluid expressed from sixteen neoplasms a diplococcus which inoculated into a rabbit produced nodular growths. As the result of the general condition provoked, the animal died of marasmus. Scheuerlen in 1887 announced that he had found as the specific cause of cancer a thread-like bacillus, and in the same year Fraenkel confirmed this discovery. Ferrero also confirmed Scheuerlen's observation, though the bacillus discovered by him occasionally entered into the spore formation. A number of other observers—Sanarelli, Landerer, Babes, and Rosenthal—also described bacilli which they had found in epithelial tumors.

The bacterial theory of the origin of cancer fell to the ground, however, for lack of experimental confirmation by inoculation. All the observers found constantly that the cocci and bacilli which they had isolated gave negative results, at least so far as regards tumor formation or the production of cancer cachexia. It has since been demonstrated that most of them were harmless saprogenic or saprophytic microbes. Scheuerlen's bacillus, for instance, was identified later as the potato bacillus, though one of the forms of the proteus mirabilis seems also to have been among the bacilli in Scheuerlen's cultures. The only positive result from inoculation was Rappin's, and the value of this is very much lessened by the fact that many micro-organisms when inoculated produce nodular growths, but these are really granulomata or pseudo-tuberculous processes, not neoplasms. The probability of malignant tumors

being due to bacteria, or, as they are called scientifically, schizomycetes,—that is, organisms which reproduce by division,—was very much lessened by the fact that none of the known bacteria ever produced anything like true tumors. Certain of them, as the bacillus tuberculosis and the bacillus of lepra, produce lesions which simulate tumors, but are very easily differentiated from them. The infectious granulomata produced by bacteria never reveal, especially on microscopic examination, the slightest tendency towards a true neoplastic process. Degeneration, not new growth, is their characteristic.

The second period in this history is that in which the coccidian theory prevailed. A number of important observations were made during this time, and, though the real cause of cancer was not discovered, the discussions evoked added notably to our knowledge of the intimate structure of malignant tumors. The observations made during this period were exact, though their interpretation was erroneous. The original error was due to the fact that Halle and Virchow and Guebler had undoubtedly found protozoa in tumors in human beings. Other observers found coccidia in human kidneys. Certain portions of the cells of malignant tumors when stained sometimes seemed to resemble coccidia in their various phases of reproduction.

Convinced that what they saw in epitheliomata and sarcomata were really coccidia, because of certain analogies with coccidium oviforme and the lesions produced by that parasite, the investigators proceeded to find in this pseudo-coccidium within the cancer-cells not only all the phases of coccidial sporification, but even the complete cycle of the formation of falciform corpuscles. What they took to be spores and spore-bearing cysts were really processes of degeneration, some of them fatty and some of them hyaline. These areas of degeneration affected sometimes the nucleus of the cells and sometimes the extranuclear protoplasm. The falciform corpuscles described by various observers were really peculiar arrangements assumed by the nuclear substance while it was undergoing degeneration.

Founded on such a misunderstanding, the coccidial theory of the origin of cancer necessarily fell a victim to the critics. It was unfortunate that, as a result, the medical world seemed to be drawn farther away from the idea that cancer was parasitic in origin.

As a matter of fact, however, the discussion of the coccidian theory and the exact observations which the study of the subject for both sides of the question necessitated have done much to introduce a scientific epoch into the study of various appearances in cancer, and this has undoubtedly brought us nearer to the solution of the problem of its genesis.

Two observers especially upheld the coccidian theory, whose descriptions and illustrations undoubtedly concern true parasitic forms and not cellular degeneration. These two are Foà and Soudakewitch. Foà described certain appearances in glandular epitheliomata which seem surely to have been parasitic in character. In the fresh specimen these parasites are seen surrounded by a capsule occupying the protoplasm and never the nucleus of the cell. Young forms can be demonstrated in the form of corpuscles slightly colored with hæmatoxylin; these sometimes appear uniform and at other times are more or less granular in the centre. Foà also saw other parasitic forms larger than those just described, consisting of a central nucleus surrounded by a clear space, on the outside of which a capsule could be traced, so that, all in all, the corpuscle had a somewhat cystic appearance. At times the parasites were pressed close to the nucleus. They were of about the size of a small white blood-corpuscle, and their capsules took the stain intensely, though at times fine striations could be seen. Occasionally a species of regular segmentation could be seen, somewhat recalling a rosette. Usually the cystic forms prevailed. These consisted of rather large-sized bodies surrounded by a capsule of double contour. The capsular contents at times took on a blue color, and several smaller bodies of unequal size but of regular configuration could be seen within them. These seemed to be due to a uniform segmentation of a primitive single central corpuscle. Other types of the parasite were marked by the presence of a number of very minute little bodies in their protoplasm. These bodies were composed of a central granule surrounded by a clear space and by a very thin capsule which was easily stained.

Whether the very diverse appearances that Foà described were due to a variety of parasites, or, with much more probability, to various phases of the development of a single parasite, was a question which Foà did not feel himself capable of solving. He considered that the regular segmentation of the protoplasm in certain

cases, which gave to the corpuscle the appearance of a rosette, represented a special stage in the development of the parasite. This regular geometric segmentation, with the regular striation of the capsule, could point only to some such process as has been described for malarial parasites. As the malarial parasite is itself a protozoan, this has all the weight of probability. The parasitic forms with included cystic appearances and granular contents would seem to indicate the formation of spores of the organism.

One observation of Foà's was particularly interesting. He was the first to call attention to the distribution of parasites in malignant tumors and to point out where they could reasonably be expected to occur if they were present. At the centre of the tumor degeneration has always taken place, and, as the body-cells have become unrecognizable, so the parasitic cells near them would be very apt to suffer in the same way. At the periphery of the tumor parasites, if present, should be found most frequently and in their most vigorous forms. This observation of Foà's has been fully confirmed by other observers studying parasites in cancer.

Soudakewitch, after a series of careful observations, came to conclusions similar to those of Foà with regard to the presence of parasites in cancer. Having examined ninety-five malignant tumors, he believed himself in a position to announce that in every case he had been able to find intracellular parasites belonging to the class of the sporozoa. According to him, the presence of the parasite always determined an hypertrophy of the cell and at times also a modification of its protoplasm. Changes in the nucleus also took place, often in the direction of karyokinesis, showing that the irritation of the cell led to a resumption of the embryonal quality of division.

The observations of Foà and Soudakewitch were perfectly in accord and were confirmed by a number of other observers. Nevertheless, the opponents of the parasitic theory of the origin of cancer insisted that the appearances described were solely the result of the degeneration of the nucleus and of the cell. This was, however, the only point on which the opponents of the theory agreed among themselves. No two of them were in accord as to what particular species of degeneration caused the appearances which had been taken for coccidia. Some thought that it was fatty degeneration, others colloid, others hyaline, still others horny, others

mucous, still others keratinic. Other degenerative processes were also invoked to explain them,—abortive forms of karyokinesis with special degeneration, hydropic degeneration, hyperchromatosis and hypochromatosis, vacuolization, and still others.

The third period of the history of parasitism in cancer begins with the labors of Russell in England in 1880 and continues and ends with those of Banti and Nisser in 1894. Russell described the parasitic cause of cancer as spherical bodies absolutely homogeneous and without visible internal structure, surrounded by a clear space which takes stains very feebly. These parasites are disposed in the cancer tissues in groups containing several individuals. They are found sometimes in leucocytes, but more usually in the cells of the tumor itself or in the connective tissue of the tumor. They multiply by budding, like the budding fungi of Naegeli. Banti encountered the same bodies in Paget's disease, which is well known to precede cancer for many years and to end almost invariably in malignancy. Banti did not attribute any serious etiological importance to these bodies. Finally Nisser succeeded in isolating from the blood of a female patient suffering with epithelioma of the uterus a blastomycete, that is, a unicellular organism resembling a ferment. He obtained this parasite in pure culture, and at once announced that it was probably the cause of the malignant tumor. This was the first advance into a new era in the study of the etiology of cancer. Nisser did not follow up his discovery, but it was too pregnant with suggestiveness to be permitted to drop from sight.

The fourth period in the history of the etiology of cancer begins with San Felice's and my own researches into the pathogenic action of blastomycetes on the human system and their relation to the etiology of cancer. In January, 1895, San Felice announced that he had isolated from the atmosphere a blastomycete which inoculated into animals caused the formation of nodular tumors. By a special method of staining he was able to demonstrate in these tumors certain appearances which resemble what had been considered to be coccidia in human cancer. On February 20 of the same year I published a first memoir on the presence of blastomycetes in adenocarcinomata of the ovary. I described and illustrated special parasites lying between the cells in sections of the tumors. The appearance of these parasites, when stained by the method of San Felice,

with certain improvements in the technique introduced by myself, their chemical reactions, and their morphology pointed to the fact that they were of vegetable origin and belonged to the class of organized ferments.

After the discovery thus made of the parasitic origin of ovarian adenocarcinoma, I demonstrated, from the descriptions and illustrations of others who had written on the subject of parasites in cancer, that all the forms described by various authors as coccidia or sporozoa possessed the characters of blastomycetes. I felt myself in a position then to conclude that what these various authors had seen were really parasites, but that the observers had all made an error in the classification of the micro-organisms which they had detected. I afterwards demonstrated the same forms of blastomycetes in other malignant tumors and in their metastases.

These blastomycetes, that have been discovered in various forms of malignant tumors, resemble very much the ordinary ferments which are capable of producing alcohol in saccharine solutions and of performing the other chemical transformations which are included under the term fermentations. It seems difficult to believe that so simple and seemingly harmless a micro-organism as one of the near relatives of the family of ordinary ferments should cause so hideous and fatal an affection as is a malignant tumor. On the other hand, it must be remembered how common malignant tumors are all over the world. There is no place, so far as we know, whose inhabitants are free from this dread scourge of malignant disease. No matter where in the world a sugar solution is exposed to the air, ferments find their way into it and produce the characteristic changes that lead to the formation of alcohol. It would not be surprising, then, if a member of the same family should prove to be the cause of cancer. At least the element of ubiquity, so necessary as a trait of the micro-organism which must cause cancer, could very well be assumed to exist for a cancer ferment as it does for the ordinary saccharomyces,—that is, the usual alcoholic ferment.

After the publication of his first note, San Felice, in a series of important articles, gave an account of his further researches into the nature and effects of what he called the *Saccharomyces neoformans*,—that is, the ferment which causes the production of neoplasms. He showed that, while this ferment occasioned in

certain animals a diffuse infection with tumors in various organs, most of them composed of an enormous accumulation of parasitic cells rather than of tissue elements, in the dog it produced at times localized tumors which gave mestastases to various organs that resembled very closely in their structure the malignant tumors of man. In his third memoir, on the pathogenic action of this ferment upon various animals, he says that he found in the mammary glands of dogs masses of cells closely crowded upon one another, sometimes arranged in columns surrounded by connective tissue, an arrangement which recalls distinctly the structure of certain carcinomata and sarcomata. While in other animals the blastomycetic cells themselves are very numerous, in the dog they are not. In new formations in the dog their location is never in the centre of the neoplasm, but at its periphery, as is observed in the case of tumors in man. He notes, moreover, that in successful inoculations the lymphatic glands nearest the resultant tumors became filled with elements bearing an appearance very similar to the cells which constitute the main mass of the tumor. In the kidney, for example, metastatic nodules were formed of cell-elements resembling those of the original tumor in the mamma. Inoculations of saccharomyces neoformans into the mammæ of dogs always induced cachexia which proved fatal in less than ten months, tumors of an undoubted-epithelial nature growing in the mean time in the mammary glands. San Felice found, in cancers of the uterus and of the human lip and in epithelial tumors in cattle, a blastomycete which he succeeded in isolating in pure culture and which investigation showed to be pathogenic for other animals.

Maffucci and Sirleo, while studying the pathogenic property of a ferment called by them saccharomyces niger, found that the lesions produced by this ferment in animals, although they have a tendency to regression rather than to progression, have, nevertheless, all the characters of a neoplasm. They do not resemble at all a purely inflammatory process, and can be very readily differentiated from ordinary granulomata by the appearance of their tissues.

These researches have been confirmed by experiment and histological investigation. Kahane isolated blastomycetes from cancers of the uterus. Curtis obtained from a myxosarcoma a ferment which he called saccharomyces subcutaneus tumefaciens. When

this was inoculated into animals, it produced tumors very analogous to those of the man from whom the ferment had been isolated. Corselli and Frisco found in a human lymphoma a ferment that proved to be pathogenic to a dog. By its inoculation they succeeded in producing a pathological process very similar to that of the patient from whom they obtained the original ferment. Pianese also obtained blastomycetes pathogenic to animals from an epithelioma of the female breast. A number of observers have found in various malignant tumors ferments, but inoculation of animals therewith gave only negative results.

Ferments exist so commonly in the air that it might be supposed that their presence in many of these tumors is really due to some fault of technique in the preparation of cultures or of specimens. It might even be thought that in certain of the external cancers, as, for instance, those on the lips or in the mammæ, ferments happened for a time to grow in the secretions though they had nothing to do with the etiology of the neoplastic process. It should be remembered that, as stated in my first communication on the subject of ferments in malignant tumors, the growth in which the blastomycetes were originally found was an adenosarcoma of the ovary. From the protected situation in which this tumor developed the presence of the ferment could not have been due to secondary infection from the air. In a sarcoma of the brain recently operated upon I have had occasion once more to find blastomycetes present in pure culture. In the brain, of course, quite as much as in the ovary, aerial infection is out of the question.

We are, then, in a position to draw certain conclusions with regard to the relations between parasites and cancer.

First, in the malignant neoplasms of man and animals there occur within the cells and in the connective tissue certain bodies that are not due to cell degeneration, but are derived from something extraneous to the animal tissues.

Second, these bodies are morphologically identical with the so-called coccidia which have been described by various observers as inclusions within the cells of epithelioma and sarcoma.

Third, the bodies which occur in cancer are also morphologically identical with certain blastomycetes which can be made to produce tumors in the tissues of animals by experimental inoculation. The principal precaution to be taken in the inoculation is to make sure

that the ferments are present in the inoculated material in pure culture.

Fourth, the bodies found in human tumors resist acids and alkalies in the same way as do the blastomycetes which exist in the tissues of animals as a consequence of inoculation.

Fifth, these bodies occur only in malignant neoplasms,—never in any other pathological process. They are always found at the periphery of the neoplasm,—that is, at the parts particularly concerned in the growth of the tumor,—never at its centre, where growth has ceased and the cellular elements are in process of degeneration.

Finally, these bodies, which can be obtained in pure culture from the malignant neoplasms of man and of animals, are blastomycetes. When inoculated into animals, these blastomycetes penetrate into the cells of the tissues and work their way in between the fibres of the connective tissue of the parts. Here they reproduce the same forms as originally existed in the tumor of the human being or other animal from which the blastomycetes were obtained. San Felice and myself have shown that in some animals experimentally infected with blastomycetes lesions are produced which are essentially of a neoplastic and not of an inflammatory character. San Felice has shown that among the higher mammals, as the dog, not only may blastomycetes after inoculation produce a tumor at the site of infection, but that this tumor may afterwards by means of the lymphatics spread into various organs, ultimately producing the death of the animal by cachexia. This fact—the production of general metastases—is most interesting and convincing.

In a word, though not all the difficulties in the important question of the etiology of cancer are yet solved, a distinct step of sure progress has undoubtedly been made. Once the cause is known, it is to be hoped that we shall be in a position to combat more effectively than ever before this dread disease, which now causes as great a mortality in the human race as most of the infectious diseases put together. For plague, cholera, and small-pox, and even typhoid fever, are as nothing in their fatal ravages in comparison with this hitherto mysterious scourge of mankind.

(See frontispiece for illustrations showing the blastomycetes.)

Laboratory Methods

THE PORTABLE LABORATORIES OF THE MARINE HOSPITAL SERVICE.[1]

BY H. D. GEDDINGS, M.D.,
Passed Assistant Surgeon, M.H.S.

Possibly prior to, but certainly ever since the early part of the year 1893 the desirability of a portable bacteriological laboratory has been recognized by the Marine Hospital Service. Charged, as this Service is, with preventing the introduction and dissemination of contagious and infectious diseases in the United States, it has frequently been called upon to investigate reported outbreaks of such diseases in widely separated parts of the country. Notably such instances have presented themselves in a reported outbreak of a suspicious disease simulating Asiatic cholera at the Arkansas penitentiary, at Little Rock, Arkansas, in January, 1893; again, in 1894, a Russian immigrant died at Cumberland, Maryland, under circumstances which led to the suspicion of the same disease; a case of Asiatic cholera was also diagnosed and isolated in Jersey City, New Jersey. On more than one occasion in the immediate past cases of disease resembling bubonic plague have been the subject of clinical and bacteriological investigation by officers of the Marine Hospital Service at the quarantine stations of the United States.

Sometimes, in the larger cities, it is possible to make use of the pathological and bacteriological laboratories connected with the medical colleges. But it is not always agreeable to present oneself in the attitude of a suppliant for laboratory accommodations, espe-

[1] Prepared under the direction of the Surgeon-General, M.H.S.

cially when engaged in the investigation of diseases which are notoriously dangerous and naturally dreaded.

But such facilities can seldom be obtained. Infectious or contagious diseases may first occur at some spot remote from educational centres, where the local practitioners of medicine, however courteous and well disposed, possess no laboratory facilities whatever, and where, as in the case of many small villages, ordinary microscopic apparatus may not be available, to say nothing of gas, running water, and other conveniences which are essential to a well-conducted laboratory.

The matter of a portable or travelling laboratory, therefore, has received the careful consideration of the Marine Hospital Bureau. A portable laboratory, constructed in 1896, was used on several occasions, but the outfit therein contained was small and incomplete. The subject was revived in the latter part of 1899, and was brought to the attention of the Surgeon-General by the writer and Passed Assistant Surgeon M. J. Rosenau, Director of the Hygienic Laboratory of the Marine Hospital Service. Our suggestions met with ready acquiescence by the Surgeon-General, and we were authorized to prepare plans for a complete portable travelling laboratory. This promised to be of immediate value in view of the threatened invasion of the United States by the bubonic plague.

There are certain desiderata which a travelling laboratory should possess. First, it must be strictly portable; second, it must be complete in all of its appointments; third, it must be so arranged and packed that any piece of apparatus or particular line of apparatus may be reached without unpacking the whole; fourth, it must be so arranged that pathological and bacteriological investigations can be pursued where there is neither gas, running water, nor other laboratory convenience; and, fifth, while complete in all of its appointments, its weight and bulk must not be excessive, nor must any one package be of such dimensions or weight as to render it difficult of transportation either by railroad or wagon or other conveyance over rough country roads.

These requirements, it is believed, have been met in the portable laboratory designed by the Marine Hospital Service. It has been tried in practice, and was exhibited at the meeting of the American Medical Association at Atlantic City, New Jersey, in June, 1900.

The apparatus is diversified and adequate, constituting, in fact, a complete laboratory so packed that the whole or any part of it may be erected at any point and work begun within half an hour after its receipt.

To show the completeness of the outfit the general contents of the nine boxes which it comprises will be here enumerated in brief.

Box No. 1 contains an Arnold steam sterilizer of the well-known Boston Board of Health pattern and of the usual size.

Box No. 2 contains an improved dry-air sterilizer with sheets of asbestos for the protection from radiated heat of the box or table upon which it may be placed. Both of these sterilizers can be heated by gas if obtainable; if not, oil heaters are provided. Box No. 2 also contains a full line of graduated glass-stoppered cylinders, with capacity of from twenty-five to one thousand cubic centimetres.

Box No. 3 contains five drawers arranged after the manner of a bureau. In these there is an assortment of small glassware, a large number of test-tubes already plugged with cotton and sterilized, flasks of all of the ordinary culture media, as agar-agar, gelatin, agar gelatin, bouillon, blood-serum, etc. Petri plates are supplied in large number. The box contains also pipette stoppered bottles for stains, the stains themselves being in other bottles, inoculating needles, hypodermic syringes for animal inoculation, Sternberg bulbs for the collection of blood and fluids, and various small apparatus for the investigation of urine, blood, and other fluids of the body.

Box No. 4 contains general laboratory supplies such as rubber tubing, wire gauze, filter-paper, cork-borers, and, in addition, a complete outfit of such cooking utensils as are in ordinary use in a bacteriological laboratory for the preparation of media. It also contains anaërobic apparatus for the investigation and cultivation of such pathogenic organisms as may do best under anaërobic conditions, a complete post-mortem case, and other small articles too numerous to mention in detail.

Box No. 5 contains all of the heavy hardware and metallic apparatus which are of general utility in a laboratory and which present ordinarily such difficulties in packing and transportation. Among these may be mentioned large coal-oil burners for heating the sterilizers, a paraffin bath for embedding and mounting pathological

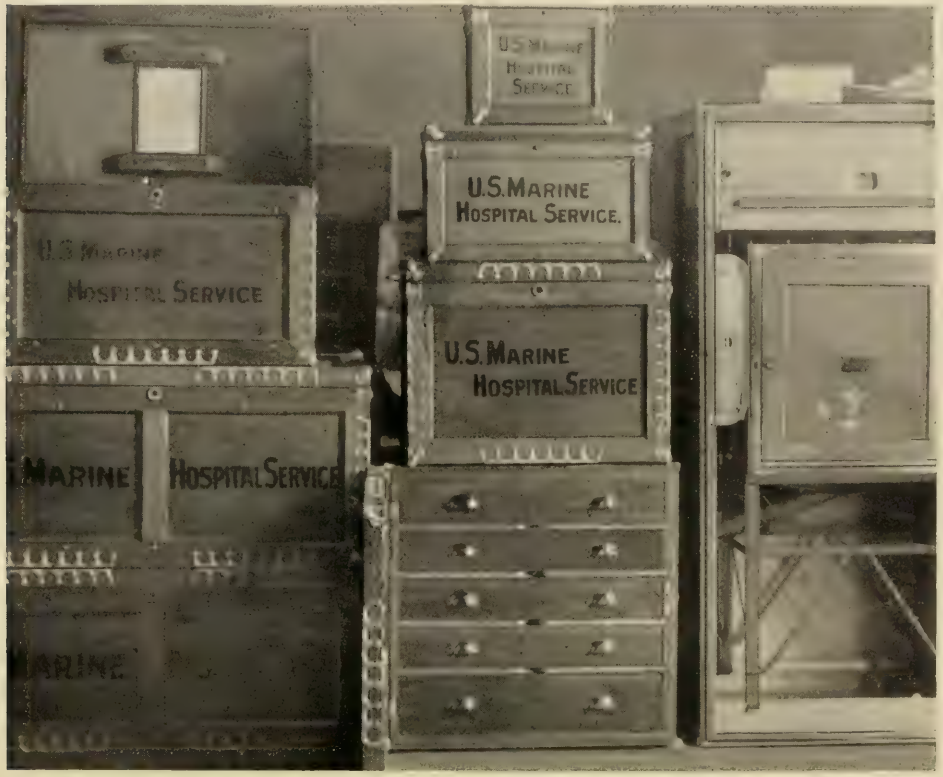

FIG. 1.—The portable laboratories of the Marine Hospital Service, packed and ready to be shipped, with the exception of the package containing the sterilizer, the lid of which is not shown in the photograph.

material, filter and funnel stands with all the usual and desirable attachments and adjustments, and alcohol blast lamps for the extemporaneous manufacture of small articles of glassware. In fact, in this box will be found almost all of the cumbrous articles required in a pathological laboratory.

Box No. 6 contains a complete incubator of the most desirable form and pattern, capable of being maintained at a uniform temperature by either gas or petroleum. This incubator is neither of the very largest size nor is it one of those small, cramped apparatus which are advertised in catalogues as being intended for use in the physician's office. It is of ample size and would furnish abundant incubating room for one or two investigators in any line of work.

Box No. 7 contains a Schanze microtome complete in all of its appointments. As this instrument is packed alone, this case need not be opened unless the necessity for section work should arise.

Box No. 8 is regarded deservedly with particular pride by the designers of the portable laboratory. It contains one of the largest and most improved of Zeiss's justly celebrated microscopes; a large microphotographic stand of the Zeiss pattern, with all recent improvements, a full line of apochromatic objectives ranging from the sixteen-millimetre (two-thirds inch) to the two-millimetre (one-twelfth inch) of 1.40 numerical aperture, and the most improved "swing-out" condenser provided with an iris diaphragm.

Box No. 9 is exclusively devoted to chemicals, and contains in moderate quantity a complete line of those reagents which are in daily use in bacteriological and pathological laboratories: paraffin for the embedding and sectioning of diseased tissues, and a very complete assortment of aniline dyes in substance. Solutions of these dyes are contained in the large stock bottles previously mentioned as a part of the contents of Box No. 3.

The total weight of the outfit when packed is about fourteen hundred pounds. Each case is of the most substantial construction, with dovetailed corners, protected by battens of hard wood, these battens being secured at the corners and protected from injury along the edges by metal strips or guards of light but substantial construction. The boxes are furnished with substantial locks. The covers are further secured by screws which work through and into metal sockets.

A conspicuous feature of the outfit is the absence of ordinary

packing material such as hay, straw, sawdust, "excelsior," and the like. Each piece of apparatus is firmly secured in its appropriate box or case by guides and specially designed clamps, adjusted by thumb-screws.

The entire apparatus was shipped by express from Washington to Atlantic City, New Jersey, where it was unpacked and displayed. During the exhibition, notice having been received of the occurrence of a case of disease simulating bubonic plague at the Reedy Island Quarantine Station, it was repacked and shipped to that point, portions of it were there used, and the entire outfit was then reshipped to Washington, where it arrived without damage to any piece of apparatus, two small flasks constituting the entire breakage.[1]

In addition to the ordinary bacteriological outfit supplementary apparatus has been added to enable the investigator to study toxin production by micro-organisms.

A laboratory complete in all of its appointments and identical with the above in its principal features, but differing somewhat in the method of packing, was sent to Manila in May, 1900, for use in the Philippine Islands.

For the sake of completeness, an itemized list of the contents of each box is herewith appended.

CONTENTS OF THE PORTABLE LABORATORY.

Box No. 1.

1 Arnold sterilizer.
2 Berkefield filters, complete.
2 culture-flasks (Miquel's), large.
2 " " (Chamberland's), large.

Box No. 2.

1 sterilizer, dry heat.
1 piece of asbestos for above.
1 burette stand.
2 burettes.
1 cylinder, graduated, glass stopper, 1000 cubic centimetres.
1 " " " " 500 " "
1 " " " " 200 " "
1 " " " " 100 " "
1 " " " " 50 " "
1 " " " " 25 " "

[1] It has since been transported and exhibited before the American Public Health Association at Indianapolis, Indiana (Figs. 1 and 2).

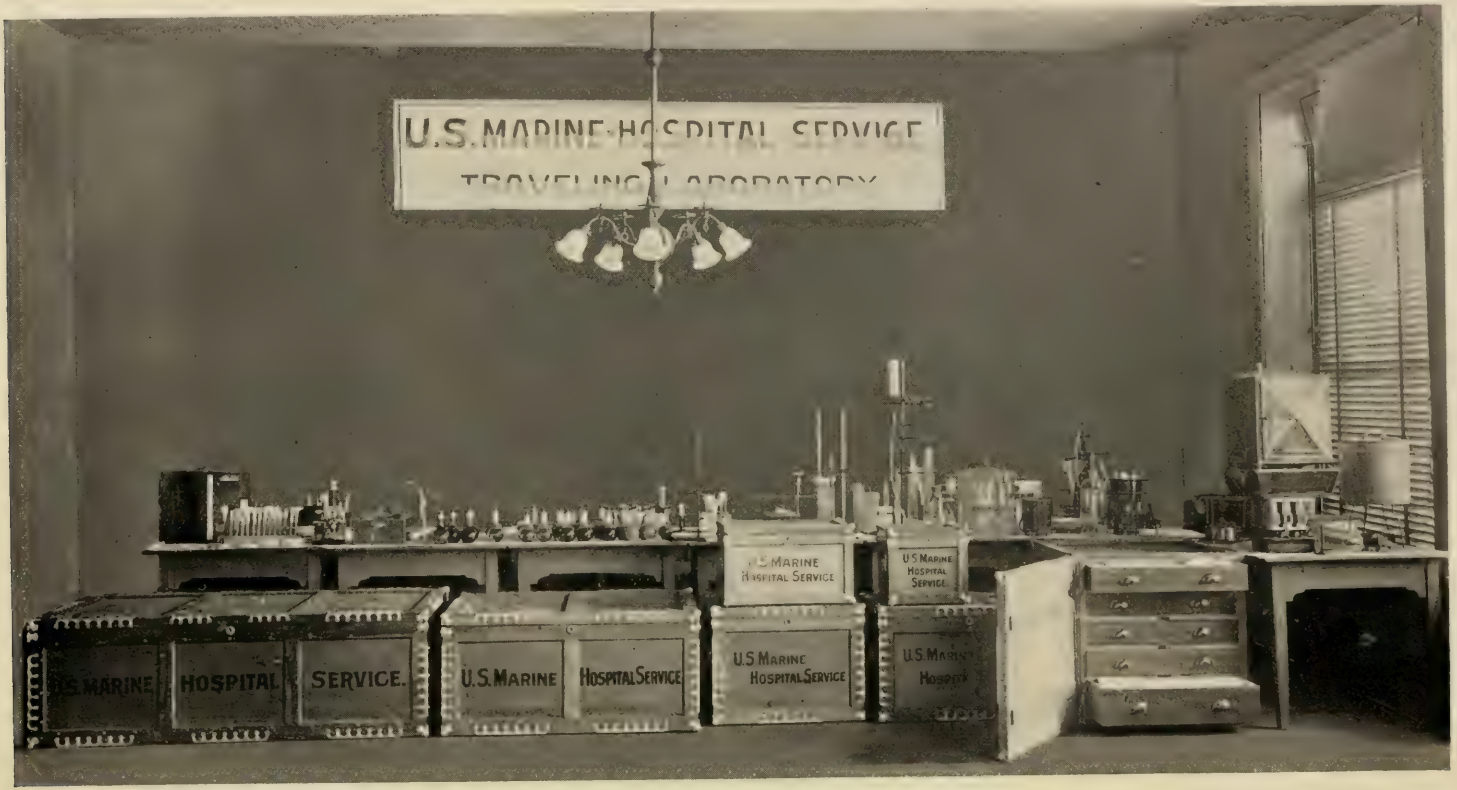
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FIG. 2.—The portable laboratory of the Marine Hospital Service as exhibited at the 1900 Indianapolis meeting of the American Public Health Association.

Box No. 3.

Drawer 1.

33 Florentine flasks, 250 c.c.
6 Erlenmeyer flasks, 50 c.c.
12 fermentation tubes.

Drawer 2.

4 culture-flasks (Miquel's).
4 " " (Chamberland's).
28 media-flasks, 250 c.c. each.
25 blood-serum tubes.
4 Pasteur pipettes.
75 Petri dishes.

Drawer 3.

2 thermo-regulators (Novy's).
24 stain bottles.
Pipettes.

Drawer 4.

12 bottles (65 c.c.), glass stoppers.
2 filter-plates (Witte's).
2 thermometers, clinical.
2 " —30° to 100° C. and F.
1 thermometer, maximum and minimum.
2 thermometers, 10° to 50° C. and F.
2 " —10° to 300° C. and F.
2 albuminometers (Esbach).
3 preparation jars.
22 test-glasses, 2 ounce.
11 " " 1 ounce.
1 platinum spatula.
2 alcohol lamps (glass).
1 ureometer.
1 pair scissors, 10 inch.
2 pairs scissors, 5 inch.
1 pair " straight, fine point.
1 " " curved, fine point.
1 " forceps, crossed serrations.
2 scalpels, wood handle.
6 wax pencils.
1 dozen test-tube swabs.
1 aspirator (Chapman's).
2 files, three-cornered.
12 pinch-cocks (Mohr's).
12 " " (Hoffman's improved).
2 gross glass slides.
3 ounce cover-glasses.
500 labels, 1″ x 1″.
4 inoculating needles.
2 oil immersion bottles.
12 watch-glasses.
1 syringe, 20 c.c.
1 " 10 c.c.
2 syringes, 5 c.c.
2 " 2 c.c.
2 " 1 c.c.
10 grammes platino-irido wire.

Drawer 5.

Sternberg bulbs.
Test-tubes, 7″ x ¾″.
" " 6″ x ½″.
7 wire baskets.
1 nest beakers.
1 graduate (glass), 250 c.c.
1 " " 100 c.c.
1 " " 50 c.c.
1 " " 25 c.c.
1 barrel glass stirrers.

Box No. 4.

In six-gallon agate stock pot.

Rubber tubing.
Wire gauze.
Filter-paper, round.
1 file, three-cornered.
1 cork-borer sharpener.
3 potato knives.
1 cork knife.
1 cork-borer set.
1 saucepan.
6 pieces asbestos, 6″ x 6″.
Rubber stoppers.
2 mortars and pestles.
1 centrifuge, complete.

In large anatomical jar.

1 anatomical jar, small.
1 Novy's apparatus for Petri plates (cock in damp chamber).

Box No. 4—*Continued.*

2 evaporating dishes, porcelain.
2 damp chambers.
6 bottles (U.S.M.H.S.), 250 c.c.
3 mouse jars.
2 syphon bottles.
3 agate dishes.
1 Novy's apparatus for tubes.
6 filter flasks, two each 1000, 500, 250 c.c.
2 Erlenmeyer flasks, 4000 c.c.
1 hydrogen apparatus, Melville jar.
1 wash-bottle.
Glass tubing.
1 gas pressure regulator (Novy's).
1 saucepan.
Filter-paper, square.
2 stands for stain bottles.
Rubber pressure tubing.
1 potato borer.

Box No. 5.

1 burner, coal oil.
1 paraffin bath.
1 water bath.
4 primus burners, complete.
1 funnel stand, large.
1 " " small.
11 rings for stands.
1 alcohol burner (Barthel's).
1 alcohol blast lamp.
1 warming table.
4 tripods.
1 burner, radial.
2 burners (Bunsen's), low form.
2 " " with regulator.
2 " " ordinary.
1 thermo-regulator (Roux's).
8 clamp adjusters.
8 clamps.
1 potato brush.

Box No. 6.

1 incubator with thermo-regulator, lamp, etc.

Box No. 7.

1 microtome (Schanze's), complete.

Box No. 8.

1 microphotographic stand with improved upper section.
1 objective, apochromatic, 16 mm.
1 " " 4 mm.
1 " " 2 mm. (ap. 1.30).
1 objective, apochromatic, 2 mm. (ap. 1.40).
1 ocular, compensating, No. 2.
1 ocular, compensating, No. 4.
1 " " No. 6.
1 " " No. 8.
1 " " No. 12.
1 " " No. 18.
1 swing-out condenser.
1 triple nose-piece.
1 mahogany case for oculars and objectives.

Box No. 9.

200 grammes acetic acid, glacial.
200 " agar-agar.
5000 " alcohol, 95 per cent.
100 " ammonium hydrate.
2 ounces Canada balsam in xylol.
100 grammes aniline oil.
200 " borax.
500 " carbolic acid.
1 ounce celloidin (Schering's).
500 grammes chloroform (Squibb's).

Box No. 9—*Continued.*

25 grammes eosin, water sol.
500 " ether.
200 " meat extract.
50 " fuchsin.
500 " gelatin.
50 " gentian violet.
200 " glucose.
500 " glycerin.
10 " hæmatin.
50 " hæmatoxylin.
500 cubic centimetres hydrochloric acid.
10 grammes iodine.
100 " lactose.
100 " litmus.
1000 " mercuric chloride.
1000 " mercuric chloride tablets.
200 " nitric acid.
50 " methylene blue.
100 " paraffin, 40 C.
100 " " 49 C.
100 " " 52 C.
100 " " 55 C.
200 grammes peptone.
200 " sodium carbonate.
200 " " hydrate.
200 " sulphuric acid.
100 " vaseline.
500 " xylol.
250 " alum.
20 " thionin.
20 " Bionde-Heidenhain.
25 " acid fuchsin.
25 " orange G.
25 " dahlia.
25 " methyl green.
2500 " absolute alcohol.
100 " oxalic acid.
250 cubic centimetres Gram's solution.
250 " " " oil origanum.
100 grammes sodium chloride.
100 " bromine.
6 pounds absorbent cotton.
500 cubic centimetres Esbach's reagent.
1 cake bichloride soap.

Monograph

THE ETIOLOGY AND MORBID ANATOMY OF VARIOUS DISEASES.

BY HENRY W. CATTELL, A.M., M.D.

The reader will here find a definition, the etiology, and the most generally accepted pathology of the more common morbid affections. The arrangement is largely alphabetical by disease, though often an anatomical classification is followed by placing the various affections of an organ under the name of the part itself. Thus, gastritis will be found under the heading of the stomach, while Addison's disease has a division of its own. The digest is based on material found in the latest edition of Ziegler's Pathology, Allbutt's System of Medicine, Guitéras's lectures delivered at the University of Pennsylvania, Osler's Practice of Medicine, and current literature, to which are added some personal observations of the author. It is hoped that this compend will be found useful for those about to pass examinations in medicine or pathology, and as a work of ready reference for those interested in these subjects. I am indebted to Doctors Yawger, Burnham, Massey, Lodholz, and Burkhard for assistance in the collection of much of the subject-matter from the above sources.

ACROMEGALY.

Definition.—A chronic disease of nervous origin, occurring most frequently in adults, and characterized by an overgrowth of the bones, especially those of the face and of the extremities, by malnutrition, and by impairment of the senses.

Etiology.—Morbid changes are always found in the pituitary body (hypertrophy, colloid degeneration, tumors, etc.) and usually in the thyroid and thymus glands. The pituitary body may be regarded as the growth-regulator of the skeleton, and the disease as primarily due to an abnormal increase in the functional activity of this gland.

Morbid Anatomy.—There are marked hypertrophy of the bones

of the face (especially of the maxillæ) and osteophytic growths on the bones of the hands and of the feet, with exaggeration of the normal ridges and tubercles. The thorax is enlarged and kyphosis may be present. The sternum is thickened, lengthened, and widened, as are also the ribs and clavicles. There may be hypertrophy of the pharynx and larynx, leading to marked dyspnœa. In one of my cases there was found after death a sarcoma of the pituitary gland; in another, all of the glands of the body appeared to be hypertrophied. I have removed *post mortem* the pituitary body through the orbit.

Under acromegaly may also be classed OSTEITIS DEFORMANS, an affection which causes softening and distortion of the long bones of the body; HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY, where there is antecedent lung disease and the bones of the skull are not involved, and LEONTIASIS OSSEA, an overgrowth of the bones of the cranium. In MICROMEGALY the condition is the reverse of that found in acromegaly.

ACTINOMYCOSIS.

DEFINITION.—A chronic, infectious disease, which occurs most frequently in cattle (as "lumpy jaw" or "wooden tongue"), but is found also in man; it is characterized by the formation of small nodules, which break down and infiltrate the surrounding tissue.

ETIOLOGY.—The *Streptothrix actinomyces* (ray-fungi) are found as yellowish opaque granules,—called sulphur balls,—which measure from half a millimetre to two millimetres in diameter. When these masses are crushed and placed under the microscope, they give the appearance so beautifully depicted (in 1856) by Lebert in his Atlas. The organism is introduced into the body with food, often through the medium of carious teeth. In one case reported the patient was accustomed to pick his teeth with a straw.

LOCATIONS.—I. Alimentary canal. II. Lungs (lesions are usually unilateral). (*a*) Chronic bronchitis. (*b*) Miliary nodules formed by masses of fungi surrounded by granulation tissue. (*c*) These nodules may run together and form abscesses and then cavities. (*d*) Bronchopneumonia. III. Thorax. (*a*) Erosion of vertebræ. (*b*) Necrosis of ribs and sternum. IV. Skin. (*a*) Subcutaneous abscesses. (*b*) Chronic ulceration, which may last for years. V. Primary infections of the brain, liver, and vermiform appendix have been described.

MORBID ANATOMY.—The characteristic primary lesion is a small nodule resembling that seen in an anatomical wart. Later there occurs, especially in the lower jaw, proliferation of cells into surrounding tissues similar to those seen in osteo-sarcoma; this is followed by suppuration. The abscesses are at first multiple, spherical, and discrete;

later they break down, run together, and give a reticulated and honeycombed appearance to the part affected. Metastases may occur.

ADDISON'S DISEASE.

Definition.—An asthenic disease characterized by loss of function of the adrenals and by pigmentation of the skin.

Etiology.—The disease is most frequently seen in laborers between the ages of twenty and forty years. It may be due to (*a*) tuberculosis, simple atrophy, cirrhosis, hemorrhage, or tumors of the adrenals. (*b*) Inflammation or pressure of surrounding structures involving the adrenals. (*c*) Changes in the semilunar ganglia and the sympathetic system.

Morbid Anatomy.—The adrenals are most frequently tuberculous, and there is always a deficiency of the internal secretion of these organs. The brownish pigmentation (bronze disease) is most marked on the chest. The spleen may be enlarged, as may also the thymus, if the latter organ persists. The stomach and intestines may show hypertrophied lymphoid follicles. No specific blood-changes have been observed. One of the most marked cases of pigmentation of the abdomen which I ever saw was that of a girl who recovered from an operation for the removal of a large dermoid cyst of the ovary. It is, of course, possible that in this case the semilunar ganglia or the adrenals were affected by pressure or otherwise. In two cases of primary sarcoma of the adrenal, and in one of general tuberculosis with marked involvement by cheesy tubercles of both adrenals, I observed no pigmentation of the skin at the time of the autopsy.

ANÆMIA, PROGRESSIVE PERNICIOUS.

Definition.—An idiopathic, chronic anæmia characterized by definite blood-changes, by a lemon-tint of the skin, by progressively developing weakness without corresponding emaciation, and by a history of "no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, and no renal, splenic, miasmatic, glandular, strumous, or malignant disease."

Etiology.—(*a*) Most common in the male sex. (*b*) Overwork. (*c*) Bad hygiene, especially poor teeth and unclean mouth. (*d*) Adult life, though the disease may occur in children. (*e*) Intestinal parasites. (*f*) Pregnancy and parturition. (*g*) Atrophy of the gastric tubules.

Morbid Anatomy.—I. *Blood.*—(*a*) Marked reduction in number of red corpuscles (to one million or less per cubic millimetre). (*b*) Alteration in their shape,—poikilocytosis. (*c*) Alteration in size,—

microcytes, macrocytes, megalocytes. (*d*) Nucleated reds,—normoblasts. (*e*) Increase of neutrophilic whites. (*f*) Hæmoglobin decreased in total amount, but ratio for each cell may be increased. II. *Miscellaneous Lesions found Post Mortem.*—(*a*) Muscles resemble horse-flesh. Heart usually large, flabby, empty, and tawny-brown. (*b*) Spinal cord may show posterior sclerosis with hemorrhagic foci. (*c*) Skin and serous membranes commonly reveal hemorrhages; these may, however, be present only in the retina. (*d*) All organs exhibit fatty changes. (*e*) Iron is deposited in excess in the lobules of the liver, especially in the outer and middle zones. (*f*) The bone-marrow—best seen in the humerus or femur, though also found in the clavicle—is red (lymphoid) in character.

ANEURISM.

Definition.—A tumor, made up wholly or in part of a circumscribed dilatation of one or more of the coats of a blood-vessel, which contains blood and which connects directly with the blood-current. (*a*) A true aneurism has a sac composed of one or more arterial coats. (*b*) A false aneurism is one the walls of which are formed by the tissues surrounding a rent in the coats of an artery. These aneurisms sometimes attain an enormous size and rupture externally in the most unexpected places. (*c*) A cirsoid aneurism is one in which the artery is twisted like a corkscrew. (*d*) A varicose aneurism has a sac communicating with both artery and vein. (*e*) Aneurismal varix results from simultaneous rupture of an artery and a vein. (*f*) In dissecting aneurism the blood circulates between the coats of the artery. I have seen such an aneurism, which began at the transverse arch of the aorta, open again into the blood-stream just above the aortic bifurcation. (*g*) Mycotic aneurisms are multiple and are micro-organismal in origin. This variety is often seen in connection with malignant endocarditis. The mesenteric arteries of the horse sometimes become dilated with considerable numbers of the *Strongylus annatus*. (*h*) Miliary aneurisms are usually multiple and consist of small dilatations; they are found especially in the brain and lungs, and often antedate a hemorrhage in these regions. They are best seen in the brain by dissecting out the middle cerebral artery and floating it in a white dish partially filled with water. In the lung they are most frequently due to tuberculous disease.

Etiology.—(*a*) Arteriosclerosis. (*b*) Strain or traumatism. (*c*) Embolic and mycotic diseases. Recent careful studies of the elastic fibres of the blood-vessels seem to show that aneurismal changes first start therein.

Morbid Anatomy.—The walls of the blood-vessels may be present or altogether absent; they may be thickened and opaque or almost transparent. If the aneurism be large, the cavity has a roughened wall, often lined with endothelium, and frequently contains clots which are white, red, organized, or softening. I have seen a fibrinous clot of an aneurism of the carotid mistaken for a sarcoma of the neck, a gluteal aneurism opened for an abscess, and a femoral aneurism mistaken for a hernia. Rupture of an aneurism, usually from the aorta into the pericardium, is a most frequent cause of death in cases brought to the notice of the coroner. The rupture often occurs during the act of defecation. Three cases of aneurism of the sinus of Valsalva have come under my notice.

ANTHRAX.

Definition.—An acute, infectious, contagious disease, more common in the lower animals (especially sheep) than in man, caused by the *Bacillus anthracis*, and having for its characteristic lesion a pustule.

Etiology.—(*a*) Certain animals are predisposed, especially sheep and goats, but the Angora sheep are apparently immune. (*b*) Certain occupations, as wool-sorting, etc. (*c*) Ingestion of the flesh or milk of an infected animal. (*d*) The *Bacillus anthracis* is the exciting cause. It is a rod-shaped micro-organism, from two to twenty-five microns in length, which is non-motile (thus distinguished from the similarly shaped but motile *Bacillus subtilis*), often united, and grows with great rapidity. Characteristic cultures may be made on gelatin plates at ordinary temperatures. The bacillus is easily killed, but the spores are very resistant. For seven successive years Ziegler was able to produce anthrax in mice by inoculations from the same piece of dry catgut which contained the spores.

Morbid Anatomy.—I. *External Anthrax.*—(*a*) Malignant pustule. At the site of inoculation appears a papule which rapidly becomes a vesicle; later a brown eschar is formed, surrounded by small vesicles and an extensive area of brawny induration. The neighboring lymphatics are swollen, tender, and hard. (*b*) Malignant anthrax œdema. This is an extensive œdema affecting the eyelids, the head, arm, and often the entire upper extremity. It may terminate in gangrene, enteritis, peritonitis, or endocarditis. II. *Internal Anthrax.*—(*a*) Thorax. Very soon after death the upper extremities, both anteriorly and posteriorly, become dark purple, the nails are blackish blue, and dark chocolate-colored fluids issue from the mouth and nose. The cellular tissues of the upper part of the chest are emphysematous and crackle on pressure. On opening the thorax these tissues are often found infiltrated with blood and a gelatinous effusion. The pleurae contain much serum

(two or three pints), the right more than the left. The pericardial fluid is also increased (six or eight ounces). The lungs are engorged with dark-colored blood. Some portions are œdematous, others harder than normal and of a darker-red color. The bronchial glands are swollen, hemorrhagic, and friable. The heart-muscle is dark colored, soft and flabby; the heart may be empty or contain dark, semifluid blood in all its cavities. The lining membranes of the heart and larger blood-vessels are stained a color varying from cherry-red to dark chocolate, according to the time which has elapsed since death. The serous membranes throughout show extravasations of blood. (*b*) Abdomen. The intestines show lesions consisting of dark infiltrated spots, about the size of a dime, with a greenish or grayish slough in the centre, which are composed mainly of anthrax bacilli situated chiefly in the lumen of the blood-vessels (Strümpell). The cavity contains considerable serum or there may be gelatinous œdema; hemorrhages appear in the serous membrane. The liver shows less change than any other organ; it may be normal. The spleen may be larger than natural or normal in size and appearance. (*c*) Kidneys. The parenchyma is gorged with dark blood, and hemorrhages appear in the capsule. (*d*) Brain and spinal cord. Extravasations of blood are discovered between the membranes and sometimes small infarcts are found. In a recent case which I had the opportunity of studying with Dr. Morton, the pustule was on the palm of the hand. The disease was contracted from bone fertilizer while working with a trowel in the garden. Early excision of the pustule, with the application of carbolic acid to the wound, was followed by recovery.

APPENDICITIS.

Definition.—An acute or chronic inflammation of the vermiform appendix.

Etiology.—(*a*) Male sex. (*b*) Early adult life. (*c*) Active exercise. (*d*) Fecal concretions. (*e*) Foreign bodies (rare); I have several times found grape-seeds in a normal appendix. (*f*) Micro-organisms,—*e.g., Bacillus coli communis* (most common), *B. tuberculosis, B. typhosus, Streptococcus pyogenes, B. influenzæ, Staphylococcus pyogenes aureus, Proteus vulgaris, B. pyocyaneus, Actinomycetes,* etc.; of these the streptococcic infection is the most acutely virulent. *Classification.*—I. *Acute Forms.*—(*a*) Simple catarrhal. (*b*) Follicular. (*c*) Suppurative or interstitial. (*d*) Diphtheritic. (*e*) Ulcerative. (*f*) Gangrenous. II. *Chronic Forms.*—(*a*) Hypertrophic. (*b*) Obliterative. (*c*) Ulcerative.

Morbid Anatomy.—(*a*) In acute forms the organ is reddish brown,

black, or greenish yellow in color. The mucous membrane is swollen, reddened, and presents hypertrophied follicles, ulcerations, or a false membrane. The whole appendix is thickened, the serous membrane red and lustreless. In the suppurative form the abscess may be small and limited to the appendix; when large the pus frequently invades the peritoneal cavity, the sac being formed by peritoneum, fibrinous exudate, and fibrous adhesions. It should be remembered that abscess formation may start outside of the appendix and there be no perforation in cases of appendicitis. In severe cases following ulcerative or obliterative conditions the abscess-cavity may contain the whole or a portion of the appendix which has been sloughed off. Ulceration following typhoid is often seen, and perforation is not unknown. In obliterative appendicitis the entire tube is thickened, firm, and stiff; the peritoneal surface is smooth or injected and may be adherent or free. It may become cystic, the contents being clear fluid or pus. The situation of the appendix varies greatly; rarely it may be found on the left side, as in transposition of the viscera, or it may be entirely absent. I have seen the tip of the appendix resting upon the pyloric end of the stomach, entering into the formation of a left femoral hernia, or lying in the sigmoid flexure in a case of ileocæcal intussusception. An abscess of the appendix may break into the rectum or vagina. Subphrenic abscess and multiple abscesses of the liver rarely result from appendicitis. Psychic disturbances sometimes follow removal of the appendix.

ARTERIES, MORBID CHANGES IN.

Definitions.—*Amyloid Degeneration.*—Usually microscopic, but may appear as starchy or wax-like bodies,—*e.g.*, sago spleen. *Atrophy.*—A general diminution in the size of arteries, best seen in stumps after operations. *Calcification; Calcareous Infiltration.*—Seen as a diffuse or circumscribed process, usually in connection with atheroma. *Fatty Degeneration.*—Usually microscopic; occurs in association with inflammatory affections; cholesterin plates and fat droplets are often found microscopically. *Hyaline Degeneration.*—Almost always microscopic; affects mostly the elastic coat and is often the beginning of an arteriosclerosis; may occasionally appear macroscopically on the heart or pleura, presenting highly refractive, glass-like patches of varying size. *Hypertrophy.*—A general enlargement, best seen in the collateral circulation after ligation of a large vessel. *Hypoplasia.*—A congenital smallness of the arteries, best seen in the aorta, where it at times causes chlorosis. *Inflammations.*—1. Acute endarteritis (proliferative or obstructive endarteritis, thrombo-arteritis). This starts with an injury to the endothelium, proliferation occurs, and an obstruction is formed in the

vessel-wall, on which a thrombus forms, partially or completely obstructing the vessel. This may terminate in absorption, suppuration, ulceration, or fibroid change. II. Chronic endarteritis. This usually follows the acute form, but is sometimes primary. It may be local (organization of a thrombus) or general (arthritis deformans). *Syphilis.*—May be local or general sclerosis; is usually a diffuse process, affecting all the coats, especially the intima. *Tuberculosis.*—Usually a local process; it may start as an internal tubercle (gray nodule) of hæmatogenous origin or as an extension from a neighboring tubercular process. I have known a cheesy peribronchial gland to ulcerate its way through the pulmonary artery and thus give rise to miliary tuberculosis.

ARTERIOSCLEROSIS.

Definition.—A hardening of the arteries, characterized by a diminution in elasticity of the vessels and marked alterations in blood-pressure. It may be local or general.

Etiology.—(*a*) Old age. (*b*) Chronic disease,—*e.g.,* gout, rheumatism, syphilis, etc. (*c*) Overwork, especially early in life. (*d*) Chronic poisoning,—by lead, arsenic, alcohol, etc. (*e*) Infectious diseases, etc.

Morbid Anatomy.—In the *first stage* there is a loss of elasticity, due to hyaline or other changes in the subendothelial coat, and the intima is thickened. The *second stage* is characterized by a thickening of the media, with proliferative changes in all the coats; this increase of new tissue gives rise to pressure on the vasa vasorum, with interference of nutrition, which leads to the *third stage.* This consists of more or less marked macroscopic changes. The vessels are hardened, firm to the touch, do not retract or close when cut, and their lumina may be smaller or larger than normal. On the intima may be seen milk-white or yellowish patches, calcareous plates, or ulceration. There may be a marked tendency to dilatation with the formation of an aneurism or to contraction with obliteration. *Special Forms.*—(*a*) Senile. Affects larger arteries most; they are dilated, tortuous, thin but stiff; often show atheromatous changes in intima. Cyanotic induration of heart, liver, and kidneys is common. (*b*) Nodular. Nodular yellowish-white projections are seen in the aorta and its branches, particularly about the orifices; sudden dilatation or aneurism may result. (*c*) Diffuse. The lesion is wide-spread and more uniform; the intima, as a rule, does not show marked naked-eye changes. Cardiac hypertrophy is constant; the kidneys are sclerosed, their capsule is adherent, cortex irregular and often cystic.

ASCITES.

Definition.—An accumulation of fluid in the peritoneal cavity.

Etiology.—(*a*) Portal obstruction, due to (1) cirrhosis of liver, (2) chronic passive congestion of liver, (3) pressure of tumors, or (4) inflammatory adhesions. (*b*) Inflammation of peritoneum,—*e.g.*, tuberculosis. (*c*) Chronic visceral disease,—*e.g.*, valvular heart-lesions. (*d*) Enlarged spleen of malaria or leukæmia.

Morbid Anatomy.—(*a*) *External Appearances.*—Abdomen usually protrudes; sides are flattened. Superficial veins much dilated with Caput Medusæ about the umbilicus, which is prominent. The œsophageal veins are often dilated, and I have seen fatal hemorrhage occur from their rupture. (*b*) *Internal Appearances.*—Serous membrane is lustreless, whitened, and thickened. Capsules of organs are often thickened and dense. The intestines are frequently matted together by fibrous adhesions, and in women the uterus and adnexa often show a similar condition. (*c*) *Ascitic Fluid.*—Usually a light-yellow clear serum, specific gravity 1006 to 1015. Contains albumin, which often forms a spontaneous clot. In cirrhosis of liver, cancer, and tuberculosis the fluid is apt to be hemorrhagic. The fluid sometimes contains chyle, sugar, or urea. I recently posted a case where capillary drainage had been practised, and another where surgical interference was used to establish a collateral circulation between various peritoneal surfaces.

ASTHMA AND HAY FEVER.

Definition.—Conditions characterized by attacks of dyspnœa, due to spasmodic contractions of bronchial tubes and air-vesicles.

Etiology.—(*a*) Spasm of bronchial muscles. (*b*) Hyperæmia of bronchial mucous membranes. (*c*) Male sex. (*d*) Begins early in life usually. (*e*) Heredity. (*f*) Certain places, odors, pollen of flowers, dust, etc.

Morbid Anatomy.—Lesions are not marked. There are hypertrophy and widening of the bronchial tubes, with thickened mucous membranes. Emphysema of air-vesicles. Chest is barrel-shaped; dorsal spine may be curved. Charcot-Leyden crystals and Curschmann's spirals are often found in the sputum.

ATHEROMA.

Definition.—Literally a small tumor occurring usually in the neck and containing a pulp-like material. As applied to blood-vessels it includes the formation of abscesses, ulcers, plates, or necrotic patches.

Etiology.—(*a*) Senile changes. (*b*) Strain and overwork. (*c*) Poisons, organic and inorganic. (*d*) Arteriosclerosis.

Morbid Anatomy.—Owing to interference with nutrition, hyaline and fatty degeneration begins in the subendothelial layer and a pulpy mass of necrotic tissue is formed, making an atheromatous abscess. When the endothelial covering is swept off, an ulcer is formed, which may become infiltrated with lime salts (atheromatous plate) or filled in with more or less degenerated connective tissue (atheromatous patch).

BERIBERI.

Definition.—An infectious disease of tropical and subtropical countries, characterized by muscular pains and weakness, a disseminated neuritis, cardiac failure, and general anasarca.

Etiology.—Little is definitely known. Various micro-organisms have been suggested. Overcrowding and a fish diet may predispose. Two types, the œdematous and the paralytic, are recognized.

Morbid Anatomy.—The special lesion appears to be in the peripheral nerves, the sheaths of which present irregular bulgings and constrictions. Atrophy of striated muscles may appear, in which case they are dry and shining, or the affected muscles, including the heart, are pale, flabby, and fatty. Evidences of general anasarca, affecting the upper extremities most, are present.

BLADDER, INFLAMMATION OF—see Cystitis.

BLOOD.

Definitions.—(*a*) *Plethora Vera.*—A condition in which all the elements of the blood are proportionately increased. (*b*) *Plethora Serosa.*—A marked increase in the watery constituents. (*c*) *Oligæmia.*—A diminished amount of blood; occurs only as a temporary condition. (*d*) *Hydræmia, Anhydræmia.*—Abnormal increase or decrease in the watery portion of the blood. (*e*) *Hæmolysis.*—Destruction of red corpuscles; occurs after burns, certain poisons, infectious fevers, etc. (*f*) *Hyperinosis, Hypinosis.*—Increased or diminished fibrin in the blood. (*g*) *Anæmia.*—A diminution in one or more of the constituents of the blood. (*h*) *Primary, Essential, or Idiopathic Anæmia.*—An anæmia, the cause not definitely known, usually attributed to the blood-making organs, and characterized by a *disproportionate* reduction in the elements of the blood. (*i*) *Secondary, Simple, or Symptomatic Anæmia.*—An anæmia due to a definite cause, as an infectious fever, and characterized by a *proportionate* reduction in the elements of the blood. (*j*) *Poikilocytosis.*—Alteration in the shape of red corpuscles (crenated, reniform, and pyriform are most common). (*k*) *Leucocytosis.*—Increase in the number of white blood-cells without al-

teration of the relative numbers of each variety. (*l*) *Leucopenia.*—A diminution in number of white blood-cells; seen most characteristically in typhoid fever. (*m*) *Lipæmia.*—Fat in the blood. (*n*) *Uræmia.*—An excess of chemical compounds, as urea, in the blood, which should be eliminated by the kidneys or other excretory organs.

BONE, DISEASES OF.[1]

Arrested Development: "Stunted Limbs."

Etiology.—(*a*) Infantile paralysis. (*b*) Anything which causes premature ossification of an epiphysis,—*e.g.,* rickets, injury, or inflammation.

Morbid Anatomy.—The bone is smaller in all its dimensions, but the joints often show the characteristic rhachitic changes.

Atrophy.

Etiology.—(*a*) Old age. (*b*) Disuse. (*c*) Constant pressure. *Classification.*—(*a*) Concentric. (*b*) Eccentric.

Morbid Anatomy.—(*a*) In concentric atrophy there is diminution in size and weight; the bone is gradually absorbed and there is a corresponding contraction of the medullary canal. It is a local process, frequently the result of ankylosis or paralysis. (*b*) Eccentric atrophy (senile) shows diminution of weight, but not of size. The compact substance becomes converted into tissue resembling the cancellous; the spaces are larger and the bony walls thinner, with consequent marked brittleness. The neck of the femur is commonly affected.

Necrosis.

Etiology.—(*a*) Pyogenic cocci. (*b*) Other infective micro-organisms. (*c*) Compound fractures. *Classification.*—(*a*) Caries, or molecular death. (*b*) Sequestrum, or death *en masse.*

Morbid Anatomy.—(*a*) Caries is usually superficial; it follows most frequently suppuration beneath the periosteum, the bone presenting an ulcerated or worm-eaten appearance. The compact substance is softer and splenization of the marrow common. (*b*) A sequestrum is the separation of a considerable portion of dead bone from the living tissues as a result of shutting off the blood-supply by thrombosis or injury. The sequestrum may be superficial, central, or total; it may be surrounded by old or new-formed bone, or by an exudate thrown out by the inflamed periosteum.

[1] Largely from Green's Morbid Anatomy.

Osteomalacia.

Definition.—A rare disease characterized by softening of the bone from decalcification.

Etiology.—Very obscure. (*a*) Female sex. (*b*) Adult life. (*c*) Pregnancy. (*d*) Lactic acid in the bones.

Morbid Anatomy.—(*a*) There is a gradual absorption of bone salts, except a thin layer next the periosteum, with corresponding increase of marrow, which becomes splenified. The bones are very light, may be cut with a knife, bend and break readily. On section the spaces contain a reddish gelatinous mass, which later becomes yellow and fatty. (*b*) The pelvic deformity is characteristic. The sacrum is pushed downward by the weight of the body, the acetabula upward and inward by the femora, thus shortening both oblique diameters.

Periostitis.

Etiology.—(*a*) Pyogenic cocci. (*b*) Infectious granulomata. (*c*) Traumatism. (*d*) Extension from neighboring structures. *Classification.*—I. Acute: (*a*) simple, (*b*) suppurative. II. Chronic: (*a*) fibrinous or productive.

Morbid Anatomy.—(*a*) Acute periostitis is generally associated with osteomyelitis; it is usually suppurative; it affects growing bones and is rare after union of the epiphyses. The exudate rapidly separates the membrane from the bone, causing stretching, occlusion, and thrombosis of blood-vessels passing into the bone, hence necrosis of superficial osseous layers. Pyæmia may occur before the abscess is opened, infective fat embolism being a result. (*b*) Fibrinous or productive inflammations commonly follow injury or syphilis. A projecting node is formed (first of new cells) which may become fibrous or calcified; rarely suppuration occurs. Calcification begins at the surface of the bone; it is a vertical process; the new tissue may at first be easily distinguished from the old, but later they become blended together. In syphilis the subperiosteal nodes show marked tendency to suppuration and superficial caries. Inflammatory thickening of bone is always due to periostitis.

Rickets.

Definition.—A disease of children, characterized by alterations in growing bones and defective nutrition of the entire body.

Etiology.—(*a*) European countries. (*b*) Large cities. (*c*) African race. (*d*) Bad hygiene. (*e*) Bad food. (*f*) Bottle-feeding. (*g*) Prolonged lactation.

Morbid Anatomy.—(*a*) In long bones, between the shaft and the

epiphysis there is a zone of proliferation from five to ten millimetres thick, irregular in outline, and soft. The bony tissue is softer and more vascular, and the periosteum strips off readily, revealing a spongy tissue resembling decalcified bone. The ends of the bones are thicker than normal and the shafts often bent. (*b*) The cranial bones are thin and atrophied in some places; in other places there are flattened swellings, especially about the forehead. The condition may resemble hydrocephalus.

Syphilis.

Classification.—(*a*) Congenital. (*b*) Acquired.

Seats.—(*a*) Skull, most common. (*b*) Long bones,—*e.g.*, tibia.

Morbid Anatomy.—(*a*) In congenital syphilis the process may be localized to the border zone between epiphysis and diaphysis; this zone has a ragged edge and a yellowish color. Later (about sixth year) the tibia may be the seat of a chronic gummatous periostitis, leading to great thickening of the bone. The "broken-nose" deformity, the result of necrosis of the nasal bone, is also common. (*b*) Acquired syphilis manifests itself usually by inflammatory changes in the periosteum, more rarely in the medulla, which show marked tendency to form bone. In the tertiary stages the gummata, especially when subcutaneous, may cause well-marked caries and necrosis, which in the skull sometimes lead to perforation; in other cases calcareous and fibroid changes occur, not only in tissues surrounding the gumma but also in the tumor itself.

Tuberculosis.

Seats.—(*a*) Bodies of vertebræ. (*b*) Ends of long bones. (*c*) Carpi and tarsi. (*d*) Phalanges. (*e*) Metacarpi and metatarsi. (*f*) Ribs. (*g*) Certain bones of the face,—*e.g.*, the turbinated.

Morbid Anatomy.—(*a*) Tubercular processes start more frequently in the bone than in the periosteum, except sometimes in the ribs, vertebræ, and phalanges; in these cases early abscesses lead to their recognition. (*b*) Primarily the process starts with a group of miliary tubercles surrounded by granulation tissue, which may become fibrous and thus terminate the process; in this outer zone the trabeculæ become thicker at the expense of the spaces, while in the centre of the process the former are undergoing absorption. Extension takes place by the formation of neighboring tubercles, the whole tending to run together, and, by shutting off the blood-supply, to undergo cheesy change. (*c*) Secondarily these areas of caseation separate as sequestra, usually small, sometimes of the size of a walnut; a whole epiphysis (*e.g.*, the head of the femur) may thus be detached. The trabeculæ of the se-

questra are thickened, showing that a chronic inflammation was present before separation occurred. The sequestra may be soft and crumbling or they may be calcified. (*d*) When the periosteum is first affected, there is rapid enlargement, which is due to formation of tubercles and may be localized in spots or extend over a large area. Erosion of the bone follows, and in turn is usually followed by an abscess, which contains a milk-like fluid, cheesy masses, and pieces of bone. (*e*) On cutting into the abscess the following structures are met with: (1) œdematous fibroid tissue containing tubercles; (2) granulation tissue, tubercles less typical; (3) a layer, chiefly epithelioid cells, which gradually becomes more and more caseous.

BRAIN AND CORD, DISEASES OF.

Abscess of the Brain.

Definition.—A circumscribed collection of pus in or upon the brain substance, with or without a pyogenic membrane.

Etiology.—(*a*) Micro-organisms,—*e.g., Staphylococcus pyogenes, Streptococcus,* diplococci of pneumonia, gonorrhœa, and cerebro-spinal fever, Bacillus coli communis, bacillus of typhoid, etc. (*b*) Traumatism. (*c*) Extension of disease from the middle ear or mastoid cells. (*d*) Septic emboli from distant foci,—*e.g.,* abscess of the liver, ulcerative endocarditis, localized bone-disease, etc. *Classification.*—(*a*) Primary (rare) or secondary (common). (*b*) Single (from extension) or multiple (metastatic). (*c*) Large (size of a walnut or an orange) or minute (then usually multiple). *Seats.*—(*a*) Cerebrum, usually in the temporo-sphenoidal lobe (most common). (*b*) Cerebellum, especially in middle-ear disease.

Morbid Anatomy.—(*a*) Acute abscesses, usually about blood-vessels; are minute, with no definite wall; contain pus mixed with reddish *débris* and softened brain matter. (*b*) Chronic abscesses, may be superficial or deep; have a pyogenic membrane; pus has a greenish tint, an acid reaction, and may have a peculiar odor depending on micro-organisms. Such cases often give rise to interesting medico-legal disputes. After surgical interference there may be complete recovery, even in cases of very large abscesses.

Anæmia Cerebri.

Definition.—A condition in which the brain is temporarily or permanently deprived of part of its blood-supply.

Etiology.—(*a*) Mechanical obstruction to the circulation,—*e.g.,* valvular heart-lesions, thrombosis, embolism, or ligation of a vessel.

(*b*) Hemorrhage. *Classification.*—(*a*) General or local. (*b*) Acute, subacute, or chronic. (*c*) Partial or complete.

Morbid Anatomy.—The membranes are pale; small arteries over the gyri are empty, though large veins are full. The brain substance is anæmic, the surface moist, few puncta vasculosa are seen, and the cerebro-spinal fluid is increased.

Aneurism of Cerebral Arteries.

Etiology.—*Classification.*—(*a*) Single or multiple. (*b*) Large or minute. *Seats.*—(*a*) Most frequent in branches of the middle cerebral artery, especially those of anterior perforated spaces. (*b*) May be cortical.

Morbid Anatomy.—The aneurisms are usually very small, multiple, and may resemble bunches of grapes. If hemorrhage occurs in basal aneurisms, the internal capsule and basal ganglia are injured, the lesion usually being extensive. On the cortex the result of hemorrhage is much less grave.

Apoplexia Neonatorum.

Definition.—A form of hemorrhage of the brain occurring in the new-born, usually the result of traumatism.

Etiology.—(*a*) Accidents during labor, from forceps, etc. (*b*) Congenital defects in blood-vessels, brain, or skull. (*c*) May result from prolonged and severe normal labor. *Seats.*—(*a*) Meninges (piarachnoid) most frequently, often bilateral, and usually at the base. (*b*) May be between dura mater and skull; is accompanied by cephalæmatoma. (*c*) May occupy the ventricles. (*d*) May occur in brain substance about basal ganglia. (*e*) Sometimes found in parietal region and Sylvian fissure.

Morbid Anatomy.—(*a*) Usually the hemorrhage is meningeal primarily, producing brain-lesions secondarily, such as atrophy and softening, by pressure. (*b*) Cortical hemorrhage is represented by a clot, which may be encysted, softened, or organized, causing more or less injury to the brain. (*c*) When the hemorrhage is between dura and skull, fracture is said to be always present.

Ataxia, Hereditary (Friedreich's).

Definition.—A form of ataxic paraplegia occurring in children and congenital in origin.

Etiology.—(*a*) Heredity, not common. (*b*) More common in males than in females. (*c*) Early life. (*d*) A specific lesion of the cord.

MORBID ANATOMY.—(*a*) There is a gliosis of the posterior columns of the spinal cord, due to developmental errors (Osler). (*b*) Talipes equinus occurs in both feet. (*c*) Lateral curvature is common.

ATAXIA, LOCOMOTOR (TABES DORSALIS).

DEFINITION.—A chronic disease of the nervous system, characterized by a sclerosis of the cord and brain, and by incoördination, with motor, sensory, and trophic disturbances.

ETIOLOGY.—(*a*) Male sex. (*b*) Adult life. (*c*) Syphilis. (*d*) Wet and cold. (*e*) Sexual excesses, etc.

MORBID ANATOMY.—(*a*) *Spinal Cord.*—Externally the meninges are thickened and adherent. Posterior roots are atrophic and of a grayish tint. Internally sclerosis of the cord begins in the posterior-root zone, involving the outer layers of posterior columns in the lumbar region. The sclerosis gradually extends inward, involving successively the columns of Burdach and Goll; when the process reaches the upper dorsal region, it is confined to the column of Goll. The cord presents a flattened appearance posteriorly, the sides being somewhat contracted. The diseased areas are firm, grayish or grayish red in color, and the whole cord is often firmer in consistency. (*b*) *Brain.*—Changes of less consequence than in the cord may be sclerosis in restiform bodies, inferior peduncles of cerebellum, and certain cranial nerves,—the oculomotor, optic, and auditory. Atrophy of the optic nerve and hemiplegia may occur. (*c*) Peripheral nerves may show degeneration or even neuritis. (*d*) In later stages occur dermapathies and arthropathies,—*e.g.,* perforating ulcer of foot, herpes, etc., Charcot's joint, etc. There may be evidences of loss of control of sphincters.

CAISSON DISEASE.

DEFINITION.—A peculiar nervous affection, the result of a sudden reduction of atmospheric pressure.

ETIOLOGY.—Occurs in bridge-builders, divers, etc., who, after working for hours under a pressure of two or three atmospheres, have suddenly returned to air of normal density.

MORBID ANATOMY.—In fatal cases there is a marked destruction of nerve tissue in the posterior columns and the posterior portions of the lateral columns, forming fatty detritus and compound granular cells.

DELIRIUM, ACUTE.

The post-mortem findings are usually negative. There may be great venous engorgement of the meninges, and the cortex and blood-vessels

may show exudation and leucocytic infiltration into the lymph-spaces and sheaths.

Note.—Careful examination of the lungs and ileum should be made in fatal cases.

Encephalitis, Acute.

Etiology.—(*a*) Acute infectious disease. (*b*) Traumatism. (*c*) Intoxications.

Morbid Anatomy.—The minutest foci of inflammation are not recognizable by the unaided eye; later stages have a pinkish appearance or are represented by clusters of small dark-red hemorrhagic foci. When suppuration follows, these areas take the form of yellowish-white patches whose tissue soon liquefies and becomes purulent.

Hæmatomyelia.

Definition.—Hemorrhage into the cord.

Etiology.—(*a*) Traumatism. (*b*) Exposure. (*c*) Convulsions. (*d*) Tumor. (*e*) Syringomyelia. (*f*) Myelitis. (*g*) Male sex. (*h*) Middle life.

Morbid Anatomy.—The cord is usually enlarged, occasionally lacerated. The blood is generally confined to the gray matter, but may escape beneath the membranes.

Hemiplegia in Children.

Etiology.—(*a*) First or second year. (*b*) Traumatism. (*c*) Embolism or thrombosis. (*d*) Congenital defect. *Classification.*—(*a*) Embolism, thrombosis, or hemorrhage. (*b*) Atrophy and sclerosis. (*c*) Porencephalon.

Morbid Anatomy.—(*a*) The results of embolism, thrombosis, or hemorrhage depend on the extent and rapidity of the formation and on location. When the process is an acute one and extensive, the results are either immediately fatal or lead to more or less extensive destruction of the brain substance. In these processes there is a tendency to softening or suppurative change. (*b*) Atrophy and sclerosis may involve a group of convolutions, an entire lobe, or even a whole hemisphere. The affected gyri are firm, hard, and atrophied, contrasting sharply with the normal tissue. They may be uniform in appearance or there may be nodular projections. In porencephalon there is loss of substance, with the formation of cavities or cysts at the surface of the brain.

Hyperæmia, Cerebral.

Classification.—(*a*) Active. (*b*) Passive.

Morbid Anatomy.—(*a*) The cerebrum is congested, the blood-vessels are somewhat distended, and petechial hemorrhages are numerous. On section the gray substance contrasts very markedly with the white; the former is of a brick-dust color; the latter shows many punctate hemorrhages. (*b*) In passive congestion the veins of the cortex are distended; the gray matter has a deeper color and its vessels are full. The gray matter shows distention of the smaller veins, which on section allow their contents to exude as drops of blood of various sizes. Excessive passive hyperæmia may result in cerebral œdema.

Leptomeningitis, Acute Cerebrospinal.

Definition.—Acute inflammation of the pia and arachnoid of the brain and spinal cord.

Etiology.—(*a*) Acute infectious fevers. (*b*) Injury or disease of the base of the skull. (*c*) Extension of disease from nose, ear, or Eustachian tube. (*d*) Pyæmia. The organisms most commonly found are the meningococcus, the pneumococcus, the tubercle-bacillus, and the cocci of inflammation; more rarely, the bacilli of influenza and of typhoid, the colon bacillus, and the gonococcus. *Classification.*—(*a*) Simple or traumatic. (*b*) Purulent. (*c*) Tubercular.

Morbid Anatomy.—(*a*) In simple or purulent meningitis the membranes are thickened, the blood-vessels dilated, and there is more or less exudation, which may be serous, serofibrinous, or purulent. The exudation may be so extensive as to cover up the convolutions. The inflammatory process is most marked in the basilar portions. It may be unilateral or bilateral. In the former the condition is due to extension from neighboring parts. (*b*) The tubercular form of the disease is usually cortical as well as basilar. It begins as a miliary tuberculosis, and in the early stages exudate is not extensive. The ventricles also may be involved and present considerable distention and softening. The ventricles seldom suffer in other forms of the disease.

Meningitis, Acute Cerebrospinal.

Definition.—An acute infectious disease, especially of early life, characterized by inflammation of the membranes of the brain, with an exudation of fibrino-purulent material, chiefly towards the base, and due to the *Diplococcus intracellularis.*

Morbid Anatomy.—(*a*) *Membranes of the Brain.*—In acute fatal cases there is intense injection of the pia and arachnoid, with a little exudate. In more chronic cases there is a formation of fibrin or of

pus, or of both; this is most marked at the base of the brain. The meninges are much thickened and opaque. The larger blood-vessels are overfilled and many of the smaller ones are obliterated. Sometimes the entire cortex is covered with a thick purulent exudate, and there may be much lymph along the larger fissures and in the sulci. In acute cases the ventricles are dilated, the appendimæ are inflamed, and the cavity may contain pure pus. (*b*) *Cranial Nerves.*—The nerves usually involved are the second, fifth, seventh, and eighth. They are often embedded in the exudate. Micro-organisms may be found in the fibrin. (*c*) *Brain Substance.*—This is softer than normal, has a pinkish color, with foci of hemorrhage and of brain softening. (*d*) *Lungs.*—Pneumonia and pleurisy may occur. The lungs are often congested, with evidences of bronchitis. (*e*) *Abdominal Organs.*—The liver is rarely altered. Acute nephritis is sometimes present, and the intestines may show swelling of the follicles. (*f*) *Skin.*—There may be rose-colored, hyperæmic spots, resembling the typhoid rash, urticaria or pemphigus, and in rare instances gangrene. (*g*) *Eye.*—Neuritis is common, and there may be acute papillitis. Purulent chorioido-iritis or even keratitis sometimes occurs. (*h*) *Ear.*—Otitis media develops from direct extension, and frequently leads to abscesses. In one of my cases the bacillus of tuberculosis was found associated with the meningococcus. In two fatal cases examined by me there was a history of traumatism, though no sign of this was found at the post-mortem. During an epidemic the domestic animals, as the goat, should be watched for signs of disease.

Meningo-encephalitis: Chronic Diffuse or Deep Chronic Leptomeningitis.

Etiology.—(*a*) Male sex. (*b*) Early adult or middle life. (*c*) Syphilis. (*d*) Alcoholism. (*e*) Certain occupations, as those of artists, navy and army officers, etc.

Morbid Anatomy.—The membranes of the brain are thickened and opaque and more or less extensively adherent to the cortex, which is torn on attempting to remove them. The convolutions of the brain are atrophied, especially in the frontal and parietal regions. The gray matter may be obscurely outlined. The white matter is firm in consistency. The ventricles are dilated and the ependyma granular; frequently there are areas of hemorrhage or softening associated with chronic arteriosclerosis. There is an increase in the cerebrospinal fluid. Usually sclerosis of the posterior columns, with involvement of the lateral, is found. There may be an extraordinary development in the lymph connective system of the brain, with a parallel degeneration and

disappearance of the nerve-elements and the axis-cylinders, and finally shrinking and extreme atrophy of the parts involved.

Muscular Atrophy, Progressive (Spinal).

Etiology.—(*a*) Male sex. (*b*) After the thirtieth year.

Morbid Anatomy.—(*a*) Macroscopically there is great muscular wasting, beginning usually in the thenar and hypothenar eminences and thence extending to the general muscular system. In marked cases the subject may be reduced "to skin and bone." Deformities and contractures result, and lordosis is almost always present. (*b*) Microscopically the muscles undergo fatty and sclerotic change, and the terminal ends of the motor nerves are degenerated. (*c*) Examination of the cord shows the anterior roots corresponding to the diseased muscles to be atrophied. Neurogliar tissues show marked increase, most conspicuous in the antero-lateral tracts. The degeneration of the gray matter extends to the medulla. Large ganglion-cells in the motor cortex may be wasted. In a case at Elwyn which I examined post mortem the diaphragm was easily seen through when held up to the light.

Myelitis, Acute.

Etiology.—(*a*) Traumatism. (*b*) Exposure. (*c*) Certain infections. (*d*) Disease of the spine. (*e*) Disease of the cord.

Morbid Anatomy.—(*a*) The cord is swollen and soft and the pia injected. On incision a diffluent fluid may escape. The distinction between gray and white matter is often lost. Hemorrhages are frequent. (*b*) Histologically the nerve-fibres are swollen, the axis-cylinders beaded, myelin droplets abundant, and corpora amylacea may be seen. The ganglion-cells are swollen, irregular in outline, and exceedingly granular and vacuolated. In the removal of the cord in these cases great care must be taken not to produce artefacts.

Myelitis from Compression.

Etiology.—(*a*) Caries of the spine. (*b*) New growths. (*c*) Aneurism. (*d*) Parasites. (*e*) Distention of central canal with inflammatory liquid or blood.

Morbid Anatomy.—Changes appear first in the white matter, the fibres of which may within six hours swell up and disintegrate.

Poliomyelitis, Acute Anterior.

Etiology.—(*a*) Early life. (*b*) Boys more susceptible than girls. (*c*) Acute infectious fevers. (*d*) Probably a specific micro-organism.

Morbid Anatomy.—(*a*) The seat of the lesion is in the part sup-

plied by the anterior median branch of the anterior spinal artery. Cervical or lumbar portions of the cord are most often affected. (*b*) In the early stages the lesion is an acute hemorrhagic myelitis, with rapid destruction of the large ganglion-cells. (*c*) The nerve-fibres of the anterior roots corresponding to the ganglion-cells destroyed break down and disappear. (*d*) Certain anterior nerve-roots are atrophied, and the muscles innervated by them waste and become fatty and sclerotic.

Sclerosis, Insular (Disseminated Sclerosis).

Etiology.—Not definitely known. Is more common in the young than in the old.

Morbid Anatomy.—(*a*) Sclerotic areas are usually small, of a grayish or whitish color, are widely distributed in the brain and cord and in the gray and white matter. (*b*) They are more abundant about the ventricles, the central canal, the pons, the cerebellum, and the basal ganglia. (*c*) The patches are firm, dry, and sharply defined from the surrounding tissue; in some cases they may be less firm and not so well defined. (*d*) Microscopically there is a marked increase of neuroglia, the medulla of the nerves is destroyed, and the axis-cylinders persist.

BRONCHIECTASIS.

Definition.—Dilatation of the bronchi, the result of various diseases of the lungs and bronchi.

Etiology.—(*a*) Some weakening of the bronchi following catarrhal inflammation (whooping-cough, measles, phthisis, etc.). (*b*) A distending force, usually coughing. *Classification.*—(*a*) Cylindrical or uniform. (*b*) Saccular, spherical, or ovoid.

Morbid Anatomy.—(*a*) Dilatation of the bronchial tubes may be local or general: in the latter case the condition is bilateral. A number of sacculi are found opening one into another; these vary considerably in size; they have smooth walls, but may in dependent portions show ulceration which is prone to set up a fatal gangrene. Sometimes the sacs are large and situated immediately beneath the pleura. (*b*) The lungs usually show marked fibroid changes, though these may be slight. The air-vesicles are sometimes emphysematous or condensed by pressure. (*c*) Adhesive pericarditis may follow extension of inflammation from pleura. (*d*) The liver usually shows chronic congestion; it may be fatty.

BRONCHITIS.

Definition.—Inflammation, acute or chronic, affecting the bronchial tubes, but not involving the terminal bronchi.

Etiology.—I. *Acute.*—(*a*) Infectious fever. (*b*) Exposure. (*c*) Irritant gases and vapors. (*d*) Extension of inflammation from neighboring organs. II. *Chronic.*—(*a*) Certain occupations (stone-cutters, etc.). (*b*) Old age. (*c*) Saprophytic and other micro-organisms. (*d*) Repeated attacks of acute bronchitis. *Classification.*—I. *Acute.*—(*a*) Catarrhal. (*b*) Suppurative. (*c*) Croupous. II. *Chronic.*—(*a*) Hypertrophic. (*b*) Atrophic or senile. (*c*) Putrid. (*d*) Plastic. (*e*) Tubercular.

Morbid Anatomy.—I. *Acute.*—(*a*) In catarrhal inflammations the mucous membrane is thickened, swollen, hemorrhagic, and in early stages covered with tenacious exudate. Later the exudate becomes thinner and purulent. (*b*) In suppurative bronchitis small abscesses, the result of septic embolism, are found in bronchial tubes. (*c*) Croupous bronchitis is characterized by the formation of a diphtheritic membrane, which sometimes forms complete casts of the smaller bronchi, but is not, as a rule, associated with the Klebs-Löffler bacillus. Bronchopneumonia is a frequent complication. II. *Chronic.*—(*a*) Hypertrophic. The whole lung is larger, firmer, and darker in color. The mucous membrane is thickened and often shows petechial hemorrhages. The whole bronchus is thicker and more fibrous; its lumen is sometimes narrowed, sometimes dilated. The surrounding lung is usually emphysematous and shows increase of fibrous tissue. (*b*) Atrophic. Often the lung appears smaller, also lighter in weight and color. Its elasticity is impaired, and it feels "cottony" to the touch. There may be increase of connective tissue, but the mucous membrane is smooth, atrophied, and the lumen of the tubules may be widened. (*c*) Putrid. This is practically a bronchiectasis. The bronchi are dilated; their walls are usually smooth, but frequently show ulcerations, with fatty plugs and purulent masses such as are found in the sputum during life. Purulent œdema of lung is more or less general. (*d*) Plastic. A chronic form of "croupous," occurs only rarely and is paroxysmal. The casts are distinctly more fibrillated and firmer than in the acute variety. (*e*) Tubercular. This may be acute, but usually manifests itself as a part of a diffuse caseous process or as tubercular ulcerations resembling those of the larynx.

CANCER OF THE BILIARY PASSAGES.

Etiology.—(*a*) Occurs most often in women. (*b*) Age forty to sixty years. (*c*) Heredity. (*d*) Gall-stones. (*e*) Parasites.

Morbid Anatomy.—Histologically it is usually a cylindrical-celled carcinoma, often primary, and generally affects the gall-bladder, which may be atrophic or dilated; gall-stones are present in most cases. The

cancer itself is usually small, flat, and ulcerating. Marked jaundice is almost invariably present. It is a debated question whether the gall-stones which are so constantly found give rise to or arise from the cancerous process. In one of my cases of primary cancer of the gall-bladder the cancerous portions and the liver had become infected by the *Bacillus pyocyaneus.*

CHLOROSIS.

Definition.—Chlorosis is a primary anæmia which occurs usually in girls, and is characterized by marked diminution of hemoglobin and sometimes by hypoplasia of the circulatory and generative organs.

Etiology.—(*a*) Female sex. (*b*) Age from fifteen to twenty. (*c*) Bad hygiene and overwork. (*d*) Shock or fright. (*e*) Absorption of intestinal poison.

Morbid Anatomy.—Cases of simple chlorosis rarely come to autopsy. The blood shows marked diminution of hemoglobin. In severe cases there may be great alterations in the number, shape, and size of the red corpuscles. The white rarely show much variation. The flesh is usually well preserved. The skin is pale and of a greenish hue, and there may be other evidences of anæmia. The internal organs will be found pale and flabby. The heart and larger blood-vessels and the generative organs show hypoplasia.

CHOLERA ASIATICA.

Definition.—An acute infectious disease originating in Eastern countries, characterized by the presence of spirochæta and by a profound inflammation of the bowel.

Etiology.—(*a*) Hot climate. (*b*) Bad hygiene and overcrowding. (*c*) The comma-bacillus of Koch. This micro-organism is a spirochæta about half the length of a tubercle-bacillus, but thicker. It may be spiral or S-shaped. Koch's bacilli are found in large numbers in the rice-water stools, but rarely in the vomit. The organism is motile.

Morbid Anatomy.—(*a*) In very acute cases the intestinal lesions are not characteristic, but the bowel contains large quantities of "rice-water" with enormous numbers of the micro-organisms. In more protracted cases the bowel presents a mapped appearance,—some areas hyperæmic and some anæmic, some hypertrophic and others ulcerated. The inflammation is well marked in Peyer's patches. The serous membrane is sticky and of a rosy color. The blood-vessels are prominent and the body looks thin and shrunken. The mesenteric glands are swollen, soft, and of a reddish color. (*b*) The stools are largely serous and contain masses of columnar epithelial cells and almost pure cul-

tures of the micro-organism. (*c*) The kidney is swollen, of a violet hue, and shows the changes of acute diffuse nephritis. (*d*) The liver shows little alteration except cloudy swelling, with minute areas of focal necrosis. (*e*) The heart is flabby. Its right side is usually distended with dark tarry blood. The left heart is usually empty. (*f*) The lungs are collapsed and show marked congestion at their bases. Pneumonia and pleurisy may develop, and abscesses are not uncommon. (*g*) There is a decided tendency to the formation of diphtheritic exudate on mucous membranes, particularly in the throat.

CHOREA, ACUTE.

Etiology.—(*a*) Female sex. (*b*) Early life (before the fifteenth year). (*c*) Heredity. (*d*) Bad hygiene. (*e*) Fright. (*f*) Bad habits.

Morbid Anatomy.—No constant lesions are found. Vascular changes, such as hyaline degeneration, leucocytic infiltration, minute hemorrhages, and thrombosis of small arteries, have been described.

CRETINISM.

Definition.—A low form of congenital idiocy.

Etiology.—(*a*) Heredity. (*b*) Special locations,—*e.g.*, Switzerland and certain places in America. (*c*) Atrophy or absence of the thyroid gland.

Morbid Anatomy.—The condition usually appears at birth. The child is stunted and dwarfish in appearance. The trunk is large in proportion to the development of the head, hands, and feet. The head is flat, the face broad and expressionless, the eyes are dull and stupid, the nose is flat and depressed, the lips are thick, and the tongue is large and usually protrudes. The teeth are carious; the hair is thin, brittle, and harsh to the touch; the skin about the hair is dry and scurfy. The abdomen is prominent; the legs are short and thick, the hands and feet undeveloped. The skin is yellow, leathery, and rough to the touch.

CYSTITIS.

Definition.—Inflammation of the bladder.

Etiology.—(*a*) Irritants in the urine. (*b*) Extension of inflammation from adjacent parts. (*c*) Traumatism. (*d*) Septic infection through the blood and urethra. (*e*) Infectious diseases. *Classification.*—(*a*) Acute. (*b*) Chronic.

Morbid Anatomy.—(*a*) In acute cases the mucous membrane is swollen, reddened, and covered with a thin film of mucus or pus. When hemorrhage has occurred, the surface of the membrane is of a universal gray tint, or mottled with gray, black, or reddish-brown patches. In

severe cases there may be necrosis, abscess, or even perforation. (*b*) In chronic cystitis the bladder may be enlarged, but it is often smaller than normal. The various coats are much thickened and there may be true hypertrophy of the muscular coat. In severe cases the inner coats often feel rough and sandy to the touch from encrusted salts.

DENGUE.

Definition.—An acute infectious (probably contagious) disease, usually found in our Southern States, generally known as "break-bone fever."

Etiology.—Supposed to be micro-organismal.

Morbid Anatomy.—Being rarely fatal, no observations have been made upon its pathological anatomy. The large and small joints are red and swollen. There is commonly a rash, but there is nothing distinctive about it. General enlargement of the lymphatic glands is not uncommon.

DIABETES INSIPIDUS.

Definition.—A constitutional condition characterized by the passage of large amounts of pale urine, of low specific gravity, containing neither albumen nor sugar.

Etiology.—(*a*) Male sex. (*b*) Early life. (*c*) Heredity may have some influence.

Morbid Anatomy.—The kidneys are enlarged and congested; their pelves are often dilated and may be cyst-like in appearance. The bladder is hypertrophied and the ureters are dilated. (Death has not infrequently resulted from chronic pulmonary disease.) Note.—Should this not be called chronic polyuria?

DIABETES MELLITUS.

Definition.—A constitutional disease characterized by the passage of large amounts of pale urine, of high specific gravity, containing sugar.

Etiology.—(*a*) Adult life. (*b*) Male sex. (*c*) Hebrew race. (*d*) Mental strain and worry.

Morbid Anatomy.—(*a*) There are no constant lesions of the nervous system, but tumors of the medulla, injury of the floor of the fourth ventricle, or sclerosis in various areas have been found. The sympathetic ganglia may be enlarged and sclerosed and a secondary multiple neuritis is not rare. Neuroretinitis is very common, and there may be hemorrhages in the retina and opacities in the vitreous. The most usual change is thickening and congestion of the membrane. (*b*) Croupous pneumonia, bronchopneumonia, and tuberculosis are com-

mon; any of them may terminate in gangrene. Fat embolism of the pulmonary vessels has been described. (*c*) The pancreas is diseased in fifty per cent. of the cases. There may be simple atrophy, pigmentary cirrhosis, cancer, calculi, cystic disease, or fat necrosis. (*d*) The spleen is usually small, pale, and soft, but may be enlarged and congested. Diffuse nephritis with fatty degeneration occurs frequently. Boils, carbuncles, onychia, eczema, and gangrene of the extremities are common. The blood generally appears normal, but may be loaded with finely divided fat which floats on the surface in a cream-like layer. The liver is usually enlarged, often congested, and abnormally firm to the touch; fatty degeneration is common. (*e*) The myocardium is pale and soft; rarely it may be hypertrophied. Advanced fatty degeneration of the muscular fibres is the characteristic change in long-standing cases of diabetes. (*f*) The urine is of high specific gravity, of a pale, somewhat cloudy appearance, always containing sugar and sometimes acetone, diacetic acid, and albumin.

DIPHTHERIA.

Definition.—An acute infectious, contagious disease, characterized by the presence of the Klebs-Löffler bacillus and of a false membrane.

Etiology.—(*a*) Klebs-Löffler bacillus is a non-motile micro-organism which, when grown on blood-serum, assumes a great variety of shapes. It is easily cultivated on albuminous media in from twelve to sixteen hours. The bacillus is very resistant, and will live for months even under unfavorable conditions. *Classification.*—(*a*) Nasal. (*b*) Pharyngeal. (*c*) Laryngeal. (*d*) Cutaneous.

Morbid Anatomy.—(*a*) The characteristic lesion of diphtheria is a false membrane, beginning early as a slightly raised, opaque, whitish-yellow spot on the mucous membrane. As a rule, it grows rapidly, becoming thicker, of a grayish or greenish hue, and firmly adherent to the underlying tissues. In the early stages if an attempt be made to remove it, it leaves behind a raw bleeding surface. In the later stages the membrane becomes less firmly adherent, soft, shreddy, and somewhat easily detached. (*b*) In nasal diphtheria the membrane may be slight in extent or may entirely block up the nasopharynx. It is apt to lead to extension of inflammation to the membranes of the brain. (*c*) In the pharyngeal form the exudate is usually first seen on the tonsils. It is apt to be very extensive and extend into the mouth, into the œsophagus, and even into the stomach. (*d*) In the laryngeal form the amount of exudate is often very great: it may entirely occlude the air-passages and extend to the lungs and the bronchial tubes, even to those of the third and fourth dimensions. (*e*) The cutaneous form is

somewhat less common: it is apt to occur about wounds, the false membrane being seldom extensive. (*f*) In nearly all cases of diphtheria there is marked inflammation of the neighboring lymphatic glands and often of the salivary glands. There is apt to be a bronchopneumonia, the sputum showing the Klebs-Löffler bacillus with the *Streptococcus pyogenes* or the diplococcus of pneumonia. (*g*) Endocarditis is extremely rare, but changes in the fibres of the heart-muscle are comparatively common. The serous membrane often shows ecchymoses. (*h*) The kidneys always show more or less diffuse inflammation, albuminuria being a constant symptom of the disease. The other organs show the ordinary febrile changes. (*i*) In malignant cases the micro-organisms may be found in the bladder and the internal organs. As a rule, however, the micro-organisms do not penetrate below the submucosa at the site of the lesion.

ELECTRIC CURRENTS OF HIGH TENSION, INJURIES BY.

Morbid Anatomy.—(*a*) Muscles show marked rigidity and may be distorted. (*b*) The skin may or may not exhibit evidences of burning or eschars. (*c*) The heart is usually flaccid, although the left side may be hard or tense. On the right side dark fluid blood is found distending both auricle and ventricle. The same condition exists in the left auricle, but the ventricle is almost empty. (*d*) Lungs show nothing abnormal. (*e*) The brain and cord are congested, but otherwise normal. The pupils are invariably widely dilated immediately after death. The blood is usually fluid, but clots have been found in the heart and large veins.

EMPYEMA.

Definition.—Suppuration occurring in the pleural cavity, usually accompanied by the presence of air.

Etiology.—(*a*) Pleurisy. (*b*) Extension of inflammation from neighboring organs. (*c*) Traumatism. (*d*) Micro-organisms, especially the tubercle-bacillus, the diplococcus of pneumonia, and the streptococcus and staphylococcus of suppuration.

Morbid Anatomy.—The pleurae are much thickened; their surfaces are irregular and covered with a yellowish-greenish exudate of varying thickness. There may be evidences of more or less extensive hemorrhage, also erosions, fistulae, or perforations. In severe cases there may be gangrenous change. The pus separates into two layers, a clear greenish-yellow serum above and a thick cream-like pus below. It has a heavy sweet odor, but is rarely fetid unless gangrene supervenes. A sterile culture suggests tuberculosis.

ENDOMETRITIS.

Definition.—Inflammation of the lining of the uterus.

Etiology.—(*a*) Gonococcus. (*b*) Extension of other inflammations. (*c*) Repeated pregnancies. *Classification.*—I. Acute. (*a*) Catarrhal. (*b*) Suppurative. (*c*) Diphtheritic. II. Chronic. (*a*) Hypertrophic (granulosa or polyposa). (*b*) Atrophic.

Morbid Anatomy.—(*a*) In acute forms the mucous membrane is red, swollen, and sodden; the discharge is profuse, stringy, and often purulent; in severe cases blood is present. Diphtheritic endometritis is rare except after typhoid fever, etc. (*b*) In chronic hypertrophic endometritis there is a hyperplasia of the mucosa, forming polypoid excrescences; the glandular structures also hypertrophy, become occluded, and form cysts of various sizes. (*c*) In atrophic endometritis the mucous membrane becomes thin and pigmented and the glandular structures disappear.

ERYSIPELAS.

Definition.—An acute contagious disease, characterized by a rash, and due to the *Streptococcus* or *Diplococcus erysipelatis.*

Etiology.—(*a*) Micro-organisms. (*b*) Traumatism. *Classification.*—(*a*) Erysipelas simplex. (*b*) Ambulans. (*c*) Phlegmonosum.

Morbid Anatomy.—In uncomplicated forms little more than an inflamed œdema is seen. At post-mortems the micro-organisms can be found in the lymph-spaces and in the zone of spreading inflammation. In severe forms the face is enormously swollen, the eyes are closed, the lips œdematous, the ears thickened, and the scalp swollen. Blebs and vesicles often appear upon the eyelids, ears, and forehead. Small cutaneous abscesses about the cheeks, forehead, and neck are common, while beneath the scalp large quantities of pus may accumulate. There is enlargement of the cervical glands, but this is masked by the œdema. *Complications.*—Infarcts often occur in the lungs, spleen, and kidneys; these are usually septic in character. Endocarditis ulcerosa is particularly common. Albuminuria is a constant complication, but true nephritis is only occasionally seen. Septicæmia, septic pericarditis, and pleuritis are of comparatively frequent occurrence.

FEVER, GLANDULAR.

Definition.—An infectious disease of childhood, characterized by marked enlargement of the cervical glands.

Etiology.—(*a*) Micro-organismal. (*b*) Age from one to ten years.

Morbid Anatomy.—Rarely fatal. The cervical glands are swollen

and softened; they seldom suppurate, nor do the adjacent skin and mucous membrane show any marked changes.

FOOT AND MOUTH DISEASE.

Definition.—An acute contagious disease, occurring most frequently in cattle and sheep.

Morbid Anatomy.—Begins as a small vesicle, which is at first clear, later becomes grayish, on the lips, cheeks, and pharyngeal mucous membranes. The vesicle increases in size from that of a dime to a silver dollar; it then bursts, leaving a shallow ulcer, with oval, circular, or irregular edges. The affected mucous membranes are inflamed, swollen, and œdematous, and there is considerable exudate.

FRAMBŒSIA (YAWS).

Definition.—A contagious disease of the skin, characterized by the presence of dirty or bright red-raspberry-like tubercles.

Etiology.—Presumably micro-organismal.

Morbid Anatomy.—Begins as a papule, usually at the site of an old wound. In a few days the papules are scattered over the body; they rapidly enlarge and become tubercles, which are usually circular in shape, and vary in size from that of a pin's head to a small apple. The epidermis splits or cracks, exposing a raw granulating surface, which rarely ulcerates. The disease tends to recovery in from two months to two years.

GALL-BLADDER—see Liver and Cancer of the Biliary Passages.

GLANDERS.

Definition.—A contagious disease occurring most frequently in horses and asses.

Etiology.—(*a*) *Bacillus mallei*. *Classification.*—(*a*) Glanders proper. (*b*) Farcy.

Morbid Anatomy.—(*a*) Glanders proper is an acute disease occurring most frequently on the mucous membrane of the nose and upper respiratory tract. Its characteristic lesion is a node or tubercle, which is at first spherical, later becomes flattened, then breaks down and presents more or less extensive ulcerations which tend to run together. The mucous membrane is swollen, is of a purplish or dark-red color, and there is considerable exudate from the ulcerating surfaces. The process may extend to the lungs, the characteristic lesion being a catarrhal pneumonia, in which the diseased areas show a marked tendency to break down, with the formation of abscesses. An eruption

of papules, which soon become pustular, frequently appears upon the face and about the joints. The cervical glands are usually much enlarged. (*b*) Chronic glanders usually occurs in the nose and is often mistaken for a chronic coryza. There are frequently ulcers about the turbinated bones. (*c*) Farcy may be acute or chronic. The acute form is of the nature of a phlegmonous inflammation at the point of inoculation. The process may be very extensive and lead to rapid suppuration of the surrounding parts. Metastasis to the surrounding tissues is common, accompanied by the formation of abscesses in the muscles. In chronic farcy localized tumors are found, usually in the skin, the subcutaneous tissue, and the muscles. These tumors break down into abscesses and may form deep ulcers. It has been described in man as a chronic specific pyæmia, characterized by eruptions on the skin and nasal mucous membranes, with frequent intramuscular abscesses.

GOITRE.

Definition.—Hypertrophy of the thyroid gland.

Etiology.—Unknown, although certain countries and localities seem to predispose.

Morbid Anatomy.—May be local or general. It is characterized pathologically by the variety of the morbid changes. In the same gland may be found cystic disease, with mucoid, colloid, and fatty changes; in some cases the enlargement may be due to marked vascular dilatation without the formation of new gland-tissue. Fibroid and calcareous changes are also common.

GOITRE, EXOPHTHALMIC (BASEDOW'S OR GRAVES'S DISEASE).

Definition.—A disease of doubtful origin, characterized by symptoms referable to the eye, the thyroid gland, and the ear.

Etiology.—(*a*) Female sex. (*b*) Early adult or middle life. (*c*) Nervous disturbances. (*d*) Disease of the cervical sympathetic (?).

Morbid Anatomy.—Hypertrophy of the gland is rarely as great as in ordinary goitre. It may be diffuse or unilateral. There is a marked increase in the number and size of the blood-vessels and an absorption of the colloid material, which is replaced by a more mucinous fluid. One or both eyes show undue prominence, probably due to increase of the orbital fat. The thymus gland is persistent and enlarged, and there is an increased amount of connective tissue in the neck. Pigmentation of the skin may be marked and simulate Addison's disease. Myxœdema may develop in the later stages. Emaciation may be extreme. Glycosuria and albuminuria are not infrequent. The heart

is usually hypertrophied; it may be dilated or even normal in appearance.

GONORRHŒAL INFECTION.

Etiology.—Due to disturbances set up by the presence of the gonococcus.

Morbid Anatomy.—The gonococcus has been found in the blood, which after death may be fluid or semi-liquid and tarry-black in color. (*a*) *Arthritis.*—The inflammation is acute, periarticular, and extends along the sheaths of the tendons. It is a synovitis which rarely becomes purulent. (*b*) *Conjunctivitis.*—This occurs most frequently in the new-born. It leads to thickening and ulceration of the conjunctivæ; erosions or entire destruction of the cornea may result. The skin of the lids may be destroyed. (*c*) *Endocarditis.*—An acute form of simple or ulcerative endocarditis, from which pure cultures of the gonococcus have been made. (*d*) The results are periurethral abscess, prostatitis, vaginitis, salpingitis, iritis, pericarditis, and pleurisy. All these lesions show a marked tendency to suppurative change.

GOUT.

Definition.—A constitutional disease characterized by deposits of uric acid or its salts in the joints of the extremities.

Etiology.—(*a*) Male sex. (*b*) English race. (*c*) Heredity. (*d*) High living.

Morbid Anatomy.—It is found most frequently in the great toe. It shows a marked tendency to involve the smaller joints, both of the feet and the hands. In acute stages there are notable hyperæmia and round-celled infiltration and diffusion into the joint. Macroscopically the joint is swollen, tense to the touch, of a purplish color, and glazed. In the chronic form the ligaments and fibrocartilages of the joint become infiltrated with chalky deposits (tophi). These deposits may be slight or may lead to enormous distortion of the joint. In some cases the skin may ulcerate through and the tophi be extruded. These deposits may be found in the cartilages of the ear, the nose, the eyelids, and occasionally the larynx. The kidneys usually show chronic interstitial nephritis, with deposits of urates, chiefly in the pyramids. Arteriosclerosis, with hypertrophy of the left ventricle, is very common. Cutaneous affections, such as eczema, are not infrequent.

HÆMOPHILIA.

Definition.—A constitutional disease characterized by a marked tendency to excessive hemorrhage from very slight causes.

Etiology.—(*a*) Male sex. (*b*) Heredity, the transmission being through the females to the males.

Morbid Anatomy.—Little is definitely known. The vessel-walls are unusually thin, brittle, and do not readily retract. In some cases the blood itself presents marked alterations. Hemorrhages have been found about the capsules of the joint, with inflammation of the synovial membranes.

HAY FEVER—see Asthma.

HEART, DISEASES OF.

Aortic Incompetency.

Definition.—A form of chronic valvular lesion in which the blood returns from the aorta into the left ventricle.

Etiology.—(*a*) Most common in vigorous middle-aged men. (*b*) May follow acute endocarditis. (*c*) Most often caused by an extension of arteriosclerosis from the aorta. (*d*) Congenital defects.

Morbid Anatomy.—(*a*) A slowly progressive sclerosis takes place, deforming, contracting, stiffening, and thickening the segments. Later calcification occurs, the segments becoming firmly welded together. A partial obstruction often results from projection of one or more of the leaflets into the blood-current. (*b*) Extensive changes are noted in the aorta, which is thickened, hardened, atheromatous (ulcers, plates, and patches), and often dilated; these changes extend to its branches, especially the coronaries, the orifices being narrowed. As a rule, there is a more or less generalized arteriosclerosis throughout the body. (*c*) Enormous hypertrophy of the left ventricle occurs early; later the enlargement becomes general. The muscle is thickened, often pigmented, and shows fatty and fibroid changes.

Aortic Stenosis.

Definition.—A form of chronic valvular lesion, in which the flow of blood from the left ventricle is obstructed.

Etiology.—(*a*) Male sex most frequently. (*b*) Past middle life. (*c*) Follows extension of arteriosclerosis from aorta. (*d*) Due to healing of an acute lesion.

Morbid Anatomy.—(*a*) Valves are usually thickened, rigid, and even cartilaginous; later they become calcified and the segments are firmly adherent to one another. (*b*) Sometimes only a thin membrane joins the segments, forming a partial obstruction. (*c*) First there is left ventricular hypertrophy, later right-sided enlargement, and finally, dilatation with pulmonary congestion.

Congenital Lesions of.

Morbid Anatomy.—(*a*) Foramen ovale; incomplete closure of; a common lesion either alone or in combination with other defects. (*b*) Pulmonary stenosis; next in frequency. Affects not only the valves, but also the conus arteriosus and frequently the whole artery is narrowed. Patulous foramen ovale; defects of the septa, and patulous ductus Botalli are generally present. (*c*) Tricuspid stenosis; found in connection with other congenital defects—is rare. (*d*) Defects in septa. Most frequent in posterior portion of anterior septum of ventricles. May occur in auricular septum. Abnormal distribution of arteries and other defects usually coexistent. (*e*) Persistence of the ductus Botalli. (*f*) Hypoplasia—failure of development. The heart is small in size dark in color and firm in consistency. The condition is associated with general lack of development. (*g*) Atresia of the aorta—rare—sometimes found in association with chlorosis of grave type.

Dilatation of.

Etiology.—(*a*) Follows hypertrophy. (*b*) Weakening of myocardium. (*c*) Resistance to emptying of cavities. (*d*) Adhesions to pericardium. (*e*) Diseases of the valves. (*f*) Diseases of the lung.

Morbid Anatomy.—Dilatation is most common on the right side and is most marked in aortic incompetency. The cavities may be very large and the walls proportionately thin. The capacity of the auricles may be trebled. The auriculoventricular rings are often dilated; the orifices of the venæ cavæ and pulmonary veins may be greatly distended; the endocardium is thickened and lustreless.

Endocarditis.

Definition.—By this term is meant inflammation, acute or chronic, of the valves of the heart.

Etiology.—(*a*) Acute infectious disease. (*b*) Chronic disease of other organs. (*c*) Chronic poisoning, as with lead, arsenic, etc. (*d*) Systemic diseases, such as syphilis and rheumatism. (*e*) Congenital defects. *Classification.*—I. Acute. (*a*) Simple or warty. (*b*) Malignant or ulcerative. II. Chronic. (*a*) Organic. (*b*) Mechanical or relative.

Morbid Anatomy.—(*a*) In the early stages of simple acute endocarditis the valves are reddened, thickened, sticky, and lustreless. The thrombi, which may be only pin-point in size, are especially found along the free margins of the valves, and more commonly on the mitral valve

than elsewhere in the heart. One of the best examples of ulcerative endocarditis that I ever met with was situated in the left auricular appendix. In some cases these thrombi extend across the valve, giving a fringe-like appearance to the part affected. Sometimes these warts may be of large size and cause considerable obstruction to the onward flow of the blood. These elevations may simply leave behind some thickening and opacity of the valve, they may be swept off and form emboli, or they may undergo organization by the formation of new connective tissue, thickening and often permanently destroying the valve. (*b*) Ulcerative endocarditis may arise in one of two ways,—either following an acute or being grafted upon a chronic valvulitis. The ulcers vary from a simple outline of tissue, with more or less irregular edges and superficial in appearance, to an extensive necrosis of the valve, the floor of the ulcer showing elevations which are either granulations with a purulent discharge or actual formation of small pustules. Ulcerative endocarditis terminates in the perforation of the valve, in actual destruction of the valve itself, or occasionally in healing by the formation of cicatricial tissue. It is almost invariably accompanied by pyæmic manifestations in other organs and tissues. Not infrequently aneurismal dilatations of the leaflets are found. (*c*) Chronic lesions of the valves may be primary, as the result of extension of aortic sclerosis or atheroma from the blood-vessels to the valve, or secondary, as the result of a healing of an acute endocarditis. In the former the lesion most frequently occurs at the aortic leaflet. There is a gradual thickening and infiltration of the valve, beginning at the base and extending to the edges, stiffening, thickening, and destroying the pliability of the valve and producing stenosis of the orifice. These changes are frequently followed by calcification. In the secondary lesion marked distortion and irregularity of the valves are frequently found; there may or may not be calcareous infiltration. There may be, as the result of these changes, stenosis or incompetence, or both. (*d*) In acute lesions of the valves the myocardium always shows swelling. The muscle is pale, somewhat flabby, and striated in appearance, and in the more malignant forms areas of necrotic tissue, varying considerably in size, are found along the distribution of the coronary vessels. These necrotic foci frequently undergo suppurative change, producing the form of myomalacia cordis which may result in rupture or aneurism of the heart-muscle or occasionally in healing by cicatrization. (*e*) In chronic valvular lesions the heart is usually enlarged, either hypertrophied or dilated; the muscular wall may be thicker or thinner than normal, of a yellowish color, often pigmented and friable; the endocardium and pericardium are thicker, pale and yellowish in

appearance, and have lost their lustre. The columnæ carneæ are thickened, sometimes contracted, and of a pale-yellowish color. The chordæ tendineæ are thickened, often contracted, and frequently agglutinated. The coronary vessels are usually thickened, hardened, tortuous, and elongated, often showing atheromatous changes. The pericardium frequently presents milk-spots, and sometimes pericardial adhesions. Mucoid and hyaline changes are not uncommon, calcareous infiltration occurs in places, and there is always an extensive fatty infiltration, especially about the bases.

Hæmopericardium.

Definition.—Blood in the pericardial sac.

Etiology.—(*a*) Aneurism of the aorta, of the cardiac wall, or of the coronary arteries. (*b*) Rupture or wounds of the heart. (*c*) Infective fever. (*d*) Poisons. (*e*) Asphyxiation.

Morbid Anatomy.—On opening the pericardium a greater or less quantity of fluid blood is found and the serous membranes are stained with hæmotogenous pigments.

Hypertrophy of.

Classification.—(*a*) Eccentric. (*b*) Concentric. (*c*) Simple.

Morbid Anatomy.—Hypertrophy is most common in the ventricles, especially the left. When the enlargement is excessive, the apex is broadened and the conical shape of the heart is lost. The myocardium is of a deep-red color, firm and resistant to the knife. It may measure from twenty to twenty-five millimetres in thickness. The right ventricle seldom shows an equal degree of hypertrophy; it may have a peculiar leathery consistency. Concentric hypertrophy occurs as a result of rigor mortis, and such a heart should be soaked in water before measurements are taken. The highest degree of hypertrophy occurs in cases of double aortic disease. Moderator bands are sometimes found in these cases.

Hydropericardium.

Definition.—An accumulation of non-inflammatory fluid in the pericardial sac.

Etiology.—(*a*) Rheumatism. (*b*) Bright's disease. (*c*) Valvular heart disease. (*d*) Any mechanical obstruction to the general circulation.

Morbid Anatomy.—Amount of fluid varies from a few cubic centi-

metres to a litre or more, when the sac is enormously distended. The pericardial sac is opaque, thickened, and often shows petechial hemorrhages. The myocardium may appear to be shrunken, and often shows considerable fatty deposit. The lungs not infrequently show the effect of the pressure of the distended pericardium.

Mitral Incompetence.

Etiology.—(*a*) Acute valvulitis, with destruction of valves. (*b*) Chronic valvulitis, contracting and shortening the valves. (*c*) Ventricular enlargement, not permitting closure of segments during the systole.

Morbid Anatomy.—Imperfect closure leads to (*a*) Hypertrophy, then dilatation of the left auricle. (*b*) Hypertrophy of the left ventricle. (*c*) Hypertrophy of the right ventricle, followed by dilatation. (*d*) Right auricle similarly involved. (*e*) Brown induration of the lungs. (*f*) Cyanotic induration of the other viscera. (*g*) Dropsical effusions, usually subcutaneous.

Mitral Stenosis.

Etiology.—(*a*) Female sex. (*b*) Early life. (*c*) Congenital changes. (*d*) Healing of an acute lesion.

Morbid Anatomy.—There are thickening and contraction of the tissues of the ring of the valve and the chordæ tendineæ. The heart is not enlarged; the left ventricle appears small when compared with the right. White ante-mortem thrombi are often found in the apex of the left ventricle.

Myocardium, Affections of.

I. Aneurism.

Etiology.—(*a*) Traumatism,—*e.g.,* stab wound. (*b*) Endocarditis, acute or chronic. (*c*) Chronic myocarditis. (*d*) Myomalacia cordis.

Morbid Anatomy.—Aneurism of the heart is most frequent in the left ventricle near the apex. It begins as a slight dilatation on the anterior surface. It then becomes a rounded tumor which may be as large as the heart itself. Occasionally the aneurism may communicate with the ventricle through a very small opening. Aneurisms of the valves are not uncommon.

II. Infarcts.

Etiology.—Thrombosis or embolism, usually of the descending anterior coronary artery. *Classification.*—(*a*) Anæmic. (*b*) Hemorrhagic. (*c*) Septic.

Morbid Anatomy.—(*a*) Infarcts are most common in the left ventricle towards the apex in the septum. Anæmic infarct is yellowish white, of the shape of a cone, the base projecting slightly above the surface. Occurring in the septum, it is sometimes cause of sudden death. (*b*) In hemorrhagic infarct the color is of a dark red. It is firm in consistency, and on section is fan-shaped. (*c*) Septic infarcts due to pyæmia vary in size from that of a pin's head to a pea, are yellowish, and on section discharge pus. They may perforate into the ventricle or the pericardium.

III. Atrophy.

Etiology.—(*a*) Congenital causes. (*b*) Senile changes. (*c*) Chronic valvular lesions. (*d*) Malignant disease. (*e*) Pulmonary tuberculosis, etc.

Morbid Anatomy.—The heart is small, the muscular wall thicker than normal, and of a dark red-brown color. The consistency is increased. Microscopically there is a decrease in the size of the muscle-cells, which show a deposit of yellow pigment and granules.

IV. Fatty Degeneration.

Etiology.—(*a*) Old age. (*b*) Wasting disease. (*c*) Prolonged infectious fevers. (*d*) Anæmia, acute or chronic. (*e*) Certain poisons, as phosphorus.

Morbid Anatomy.—(*a*) Macroscopically the heart appears large, flabby, and relaxed. It is of a light yellowish-brown color. The muscle has a streaked or patchy appearance and tears easily. The process may be a local or a general one. The left ventricle is most often affected. (*b*) Microscopically the muscle-fibres are usually seen to be diminished in size and to contain numerous granules arranged in longitudinal rows from the periphery inward.

V. Fatty Infiltration.

Etiology.—(*a*) Male sex. (*b*) After middle age. (*c*) Sedentary habits. (*d*) Anæmia.

Morbid Anatomy.—The heart is seldom much increased in size; it is often completely enveloped in fat, which is usually most marked about the auricles. Microscopically the fat is seen to infiltrate between the fibres of the myocardium.

VI. Myomalacia Cordis.

Etiology.—(*a*) Embolism or thrombosis causing infarct. (*b*) Extension of suppuration from the pericardium or endocardium into the heart-muscle.

Morbid Anatomy.—Occurs most frequently in the left ventricle towards the apex. The affected areas are at first firm and appear only as discolorations of the muscle. Later they soften, become friable, and frequently suppurate. The results are rupture of the heart, aneurism, metastatic pyæmia, or healing by scar formation.

VII. Hyaline Degeneration.

Etiology.—(*a*) Prolonged infectious fevers. (*b*) Early stages of arteriosclerosis.

Morbid Anatomy.—As a rule, the process is microscopic. Occasionally it may appear to the naked eye as a homogeneous, translucent deposit upon the endocardium or in the myocardium. In the latter case the affected fibres are swollen; the striæ are very faint or entirely absent.

VIII. Amyloid Degeneration.

Etiology.—(*a*) Prolonged suppuration. (*b*) Cachectic disease. (*c*) May follow hyaline changes.

Morbid Anatomy.—It is a comparatively rare affection of the heart. It affects the entire muscular connective tissues and the walls of the blood-vessels, but not the muscle-fibres.

IX. Calcareous Infiltration.

Etiology.—(*a*) Senile changes. (*b*) Advanced arteriosclerosis. (*c*) Old chronic inflammatory foci.

Morbid Anatomy.—Usually appears as plaques or plates of varying size, most frequently in the site of an old inflammation. In very marked cases the heart may appear to be almost completely encased in the calcareous deposit. The salts may even penetrate into the heart-muscle.

X. Fragmentation and Segmentation.

Etiology.—(*a*) Chronic disease of the myocardium. (*b*) Electricity. (*c*) Fatty degeneration. (*d*) Certain poisons, as phosphorus, arsenic, etc. (*e*) Aneurism of the heart.

Morbid Anatomy.—In fragmentation and segmentation the heart is large; it may be pale in color or deeply congested. The muscle is usually flabby, non-elastic, and easily torn. Microscopically in fragmentation the muscle-fibres are seen to be broken up or fractured. The protoplasm is distinctly granular and the nuclei frequently lost. In segmentation there is a separation not only of the bundles but also of the individual fibres one from the other. Both fragmentation and segmentation are usually localized processes.

XI. Rupture of the Heart.

Etiology.—(*a*) Embolism or thrombosis. (*b*) Suppurative myocarditis. (*c*) Fatty infiltration or degeneration. (*d*) Gumma. (*e*) Age sixty years or over (two-thirds of the cases).

Morbid Anatomy.—The rupture may occur in any of the chambers, but is most frequent in the anterior wall of the left ventricle near the septum.

XII. Infectious Granulomata.

Etiology.—(*a*) Tuberculosis is the most common cause. In the acute miliary form the tubercles are usually found beneath the endocardium of the right ventricle, but in the chronic forms the caseous or diffuse fibroid changes are usually associated with tuberculous pericarditis. These lesions are perhaps more frequent on the left side.

XIII. Syphilitic Gummata.

Very rare. They appear as soft, gray or grayish-red, dry, yellowish nodes, which may occasionally break into the cavities of the heart. Scars may be found as the result of syphilitic lesions.

XIV. Actinomycosis.

This disease may involve the pericardium and then the myocardium. It forms grayish nodules, which become fatty and yellowish white and tend to suppurate.

XV. Tumors.

(*a*) Primary tumors are rare. Sarcoma, fibroma, lipoma, and rhabdomyoma occur. Sometimes they are of congenital origin. (*b*) Secondary tumors are rather more common. They may be sarcomatous or cancerous. Large growths may interfere with the heart's action. Softening and ulceration may lead to rupture of the muscle. Thrombi readily form upon tumors which project into the internal cavities of the heart.

XVI. Parasites.

(*a*) Cysticercus. (*b*) Echinococcus.

Morbid Anatomy.—The echinococcus may lead to rupture of the heart and give rise to embolism of the pulmonary arteries.

XVII. Myocarditis.

Etiology.—(*a*) Acute infectious fevers. (*b*) Chronic poisoning,—*e.g.*, by lead, gout, rheumatism, syphilis, etc. (*c*) Extension from neighboring organs. *Classification.*—I. Acute. (*a*) Parenchymatous. (*b*) Interstitial. II. Chronic or indurative myocarditis.

Morbid Anatomy.—(*a*) In acute parenchymatous myocarditis the muscle is swollen, pale in color, striations are well marked, and the muscle is inelastic and of diminished consistency. Small hemorrhages are very common, especially in the endocardium and pericardium. Pigmentary deposits, especially of blood, are common. (*b*) In acute interstitial myocarditis the inflammation is usually septic in character and is associated with a formation of single or multiple abscesses, the latter being more common, following septic embolism of the smaller branches of the anterior coronary artery. It may result from extension from the endocardium or pericardium. (*c*) In chronic myocarditis the process may be local when it is the result of the healing of an abscess, wound, infarct, or, very rarely, a gumma, or it may be general when it is part of a general arteriosclerosis or a result of interference with the circulation of the heart itself. Probably repeated embolism of small branches of the coronary artery is the most common cause of diffuse change in the myocardium. It is a common accompaniment of all excessive hypertrophies and of brown atrophy of the heart.

HEMORRHAGES, CAUSE OF.

(*a*) Traumatism. (*b*) Acute inflammation. (*c*) Passive congestion. (*d*) Corrosive poisons. (*e*) Malignant growths. (*f*) Diseases of the vessels. (*g*) Rupture of an aneurism. (*h*) Cachectic disease. (*i*) Dyscrasias. (*j*) Nervous disturbances. (*k*) Vicarious menstruation.

Hemorrhage of the Bowel.

Morbid Anatomy.—The hemorrhage may be localized or diffuse. In the former petechial spots or ecchymoses are found on the mucous membrane, distributed over a greater or less extent of the bowel. The mucous membrane surrounding the hemorrhages may be normal in appearance or show the results of active or passive congestion. In diffuse hemorrhages the blood is free in the bowel or may be extravasated into the mucous membrane. In the former case it is brownish black or black in color and usually semi-liquid or tarry. In the latter case the extravasated blood is in slate-colored or black patches.

Hemorrhage, Cerebral.

Etiology.—The most common cause (sixty per cent.) is rupture of the lenticulostriate artery. *Classification.*—(*a*) Basilar. (*b*) Cortical.

Morbid Anatomy.—In basilar hemorrhage section of the brain substance frequently shows miliary aneurisms, which are seen as small dark bodies along the course of the blood-vessels penetrating the ante-

rior perforated spaces. Aneurism of a branch of the circle of Willis may be found. Endarteritis and periarteritis, with diapedesis and degenerative changes, are found in the cerebral vessels. At the seat of a recent hemorrhage the brain has a dark-red, softened appearance, the tissue being reduced to a coagulated or pulpy mass of detritus. When the hemorrhage has been extensive, the remainder of the brain is anæmic. The gyri are more or less flattened, from extravasated blood, and the sulci are indistinct. These hemorrhages are most common near the corpus striatum towards the outer section of the lenticular nucleus. They may be small and limited to the lenticular body and internal capsule or may break into the lateral ventricle. Ventricular hemorrhage is rare. It is usually bilateral. Meningeal hemorrhage is usually caused by fracture of the skull or rupture of a blood-vessel. The hemorrhage may be small or large. It may be above or below the dura or between the pia and the arachnoid.

Hemorrhage of the Pancreas.

This occurs most frequently in association with acute pancreatitis, *q.v.*

Hemorrhage, Pulmonary, or Apoplexy.

Morbid Anatomy.—This is a condition in which there is hemorrhage into the air-cells and lung-tissue. The lung is large, firm in consistency, dark in color, and heavy. On section there is extravasation of considerable amounts of fluid blood, usually more or less frothy. The extent of lung involved differs very greatly.

Hemorrhage into the Spinal Membranes.

Morbid Anatomy.—(*a*) Extrameningeal hemorrhage may be extensive, without compression of the cord. Rupture of an aneurism into the spinal canal may produce profuse and rapidly fatal loss of blood. There may be little demonstrable morbid change. Intrameningeal hemorrhage usually occurs in scattered areas as the result of acute infectious fevers. More extensive hemorrhages result from epilepsy, tetanus, and strychnine poisoning. Occasionally hemorrhage into the spinal meninges may ascend to the brain.

Hemorrhage from the Stomach.

Morbid Anatomy.—If the blood come from without, as from a rupture of an aneurism, the stomach presents but few changes. The blood may be fluid or clotted; it may be bright red or dark in color. Where the hemorrhage is due to actual disease of the stomach, this

blood is apt to be coffee-brown. Petechial hemorrhages in the mucous membranes are common. Ulcer of the stomach is usually associated with extensive hemorrhage from the wall of the stomach itself.

HODGKIN'S DISEASE (PSEUDOLEUKÆMIA).

Definition.—A disease characterized by a progressive hyperplasia or by changes of lymph-glands, by anæmia, and sometimes by secondary lymphoid growths in the liver, spleen, and other organs.

Etiology.—Unknown.

Morbid Anatomy.—The characteristic lesion is hypertrophy of the lymphatic glands, occurring most frequently in the cervical, next in the axillary, then in the inguinal glands, in the mediastinum, the thorax generally, and the glands of the peritoneum, especially the retroperitoneal. In the early stages the glands are moderately enlarged, soft, and elastic. They are isolated and sometimes freely movable. Later they become of an almost stony hardness and are surrounded by a dense capsule. They tend to increase in size and to run together. The capsule may be perforated and the growth invade the surrounding structures. On section the tumor appears grayish-white. It is smooth and the interior may be firm and dry or soft and juicy. Suppuration sometimes occurs when the growth reaches the skin. The spleen is usually enlarged, but rarely is greatly so. The surface section shows lymphoid tumors which are firm to the touch and vary in size from that of a pea to a walnut. The ulcers are usually small,—from the size of a pin's head to that of a cherry-stone,—and are pink or gray in color. Peyer's patches frequently show enlargement. There may be an almost general inflammatory involvement of the lymphatic glands throughout the body. The bone-marrow may be converted into a rich lymphoid tissue. The blood-changes are those of a distinct anæmia of the simple type. The careful study of a recent case would lead me to believe that the retroperitoneal region, especially near the pancreas, may be involved to a marked degree, and this too without a general increase in the glands other than in a single region where the enlarged glands are usually found. In this case the glands of the right side of the neck were markedly affected.

HYDROPHOBIA.

Definition.—A convulsive disease due to the action of the bacillus of hydrophobia on the nervous centres.

Morbid Anatomy.—No characteristic lesions are found at the post-mortem. The cerebrospinal system shows congestion of the blood-vessels. There are minute hemorrhages, most numerous in the me-

dulla. The mucous membrane of the pharynx is congested and not infrequently covered with blood-stained mucus. This is true of the larynx, trachea, and larger bronchi, also of the lungs, œsophagus, and stomach. Experiments have shown abundant virus in the spinal cord, brain, and peripheral nerves, but it has not been found in the liver, spleen, or kidneys. When a dog that is supposed to be mad has bitten a human being, the animal should not be at once killed, but permitted to live and kept under close observation until he shows unmistakable signs of rabies. He should then be killed and his body sent to a competent bacteriologist for inoculation experiments on rabbits. In three of my cases which had been diagnosed as true hydrophobia, the results of inoculation were negative, and the symptoms seem to have been due to uræmia.

INFLUENZA.

Definition.—An acute, epidemic, contagious disease, due to Pfeiffer's bacillus, and characterized by abrupt onset, great depression, and many sequelæ.

Etiology.—Pfeiffer's bacillus is found in the nasal and bronchial secretions. It is one of the smallest organisms known, non-motile, and stains well with Löffler's methylene-blue. On culture media it grows only in the presence of hæmoglobin. *Classification.*—(*a*) Respiratory form. (*b*) Gastro-intestinal. (*c*) Nervous.

Morbid Anatomy.—(*a*) Lesions of the respiratory form are those of an acute inflammation of the mucous membrane of the upper respiratory tract and bronchial tubes. Lobular pneumonia is common. It is probably due to a mixed infection. Pleurisy is more rare, but may lead to empyema. Tuberculosis is apt to be exaggerated by an attack of influenza. (*b*) In the gastro-intestinal form the inflammation extends to the mucous membrane of the stomach and the intestines. It is seldom of a severe type. The spleen is usually enlarged in this form. (*c*) In the nervous form mild degrees of meningitis and encephalitis are not uncommon. Abscesses of the brain have occurred in severe acute cases. In some epidemics accumulations of pus in the nasopharynx are exceedingly common. *Complications.*—Acute diffuse nephritis is quite frequent. Endocarditis, pericarditis, and thrombosis have been reported. Occasionally purpura is seen and also catarrhal conjunctivitis and iritis. In an autopsy on a child dying from meningitis following the grippe Dr. Kneass isolated for me the influenza bacillus.

INTESTINES, DISEASES OF.

Colitis.

Definition.—Inflammation of the large bowel.

Etiology.—(*a*) Early life. (*b*) Summer weather. (*c*) Improper foods. (*d*) Certain micro-organisms. (*e*) Poisons. (*f*) Some infectious diseases. *Classification.*—(*a*) Simple. (*b*) Membranous. (*c*) Ulcerative. (*d*) Chronic.

Morbid Anatomy.—(*a*) In simple colitis the mucous membrane is much thickened and reddened, the rugæ are prominent, and petechial hemorrhages are common. The follicles are often greatly swollen and may present minute points of ulceration. (*b*) Membranous colitis is characterized by the formation of a more or less complete cast of the intestine, usually from one inch to six inches in length, but it may extend a distance of several feet. The membrane usually appears homogeneous, but may be distinctly laminated and show deposits of fecal matter between the layers. The end of the cast may be well defined, but often shades off into a transparent gelatinous material. The outer surface is rough, sometimes sacculated; the inner surface is smooth. (*c*) In ulcerative colitis the appearances vary greatly: the ulcers may be small and numerous or they may be large in size and few in number. They may be perfectly regular in outline, but are usually irregular, with slightly undermined edges. The floor of the ulcer generally shows a somewhat sloughing bowel. In long-standing cases they are often intensely congested and tend to become transverse. Sometimes the floor of the ulcer becomes so thin as to be pushed out and form pouches. In very acute cases the mucous membrane is much reddened, highly vascular, and the surface is soft. The peritoneal coat of the bowel may be normal in appearance, but is usually red, somewhat sticky, and shows many dilated blood-vessels. Small hemorrhages are common. (*d*) In chronic colitis the bowel is often much thickened in all its coats. It may be larger in diameter. It is firm, even leathery, to the touch. The mucous membrane is hypertrophied, often much pigmented, and shows many small hemorrhages. There may or may not be ulceration.

Colon, Dilatation of.

There are four forms of this condition: (*a*) Distention from gas. (*b*) Distention due to some solid substance within the bowel. (*c*) Distention caused by an organic obstruction in front of the dilated bowel. (*d*) The so-called idiopathic dilatation.

Colon, Malignant Disease of.

This is generally a cylindrical-celled epithelioma, usually confined to a small area, where its contraction sets up an annular stricture.

Dysentery (Bloody Flux).

Definition.—Dysentery is an infectious, contagious disease, marked by the passage, with great tenesmus, of small bloody and mucous stools, and by certain characteristic lesions of the bowel.

Etiology.—(*a*) Warm climates. (*b*) Improper foods,—*e.g.*, unripe fruits. (*c*) Certain micro-organisms,—*e.g.*, *Amœbæ coli*. *Classification.*—I. *Acute.*—(*a*) Catarrhal. (*b*) Amœbic. (*c*) Gangrenous. II. Chronic. In the early stages the Bacillus of Shega is found, and in the later stages, especially where abscess develops, the amœba coli is seen. The blood of patients affected with tropical dysentery has an agglutinative reaction with the bacillus of dysentery.

Morbid Anatomy.—All the lesions of dysentery have certain points of election for the starting of the inflammatory process,—viz., the large bowel, the flexures of the large bowel, and the course of the valvulæ conniventes. (*a*) *Acute Catarrhal Dysentery.*—The mucous membrane is enlarged, swollen, and covered with tenacious blood-stained mucus. The solitary follicles stand out prominently and in protracted cases often show necrotic or suppurative change. In some cases numerous ulcers appear throughout the large bowel. In children the picture is that of an acute follicular colitis. At first glance the mucous membrane seems to be universally congested; on closer examination it is found to be more or less streaky, with bright-red pin-point areas of intense congestion. The peritoneal surface is enlarged, lustreless, and sticky. (*b*) *Amœbic Dysentery.*—In this form the amœbæ are constantly present. These are unicellular protoplasmatic motile organisms, five or six times the size of a white blood-corpuscle. They contain a nucleus and one or more vacuoles. The characteristic lesion is ulceration, which has a small external opening, with extensive undermined infiltrated edges. Sometimes these ulcers run together, forming deep sinuous tracts bridged over by apparently healthy mucous membrane. There is a progressive infiltration of the connective-tissue layers of the intestine, causing pressure upon the blood-vessels and subsequent necrotic changes in the overlying structures, so that the mucosa or the muscularis may be sloughed off *en masse* in certain parts of the bowel. In severe cases the whole of the intestine may be much thickened and riddled with ulcers, with only here and there islands of intact mucous membrane. More rarely these ulcers have but slightly undermined edges, the borders being more or less cleanly cut. In some cases there is a tendency to

purulent formations. (*c*) *Gangrenous Dysentery.*—This form is characterized by the formation of a diphtheritic membrane, which is more or less irregularly distributed; it is at first yellowish brown, in later stages becoming black or ashen-gray; in the latter case it appears as sloughs more or less easily detachable. There is thickening of all the coats of the intestine, with great interference with the blood-supply, so that in severe cases whole portions of the bowel may become gangrenous. (*d*) *Chronic Dysentery.*—In this form the anatomical changes are variable. Deeply pigmented ulcers are often present or there may be cicatrizations; again, no trace of ulceration may appear, but the entire mucous membrane presents a rough, irregular, figured appearance, in places slate-gray or blackish in color. Certain parts of the mucosa are greatly thickened and the muscular coat is hypertrophied. In some cases the solitary follicles are enlarged and pigmented. The calibre of the bowel may be reduced, but stricture is very rare. *Complications.*—(*a*) In all cases dysentery may be complicated by peritonitis, pleurisy, pericarditis, or pyæmic manifestations. (*b*) In amœbic dysentery the characteristic complication is the abscess of the liver, which is usually single and occupies the right lobe. It may be multiple, when it is apt to be distributed superficially in any or all of the lobes. It is a large solitary abscess, the wall of which is made up of broken-down, rough, shaggy liver-tissue, without any of the ordinary pyogenic membrane. The contents of this abscess vary. The outer portions are gelatinous and composed of broken-down liver-tissue, blood-pigments, pus-cells, amœbæ coli, etc. The interior is usually of an almost watery consistency, and of a brownish or reddish color. In some cases cultures made from these abscesses are sterile. In hot climates the amœbæ coli can almost always be found on microscopical examination.

Enteritis, Catarrhal.

Etiology.—(*a*) Early life, most commonly. (*b*) Summer weather. (*c*) Improper feeding. (*d*) Bad hygiene. (*e*) Irritant drugs and poisons. (*f*) Acute infectious fevers.

Morbid Anatomy.—(*a*) In the acute form the mucous membrane is generally pale and covered with mucus; the tips of the valvulæ conniventes may be deeply injected and the entire mucosa soft and infiltrated. In places the epithelial lining may be stripped off and appear in the contents as large flakes. Solitary follicles in both small and large bowel may be distinctly swollen; their centres present small erosions (follicular ulcers). Peyer's patches may also be swollen and prominent. (*b*) In chronic enteritis the mucosa is thickened. The

valvulæ conniventes are prominent and usually markedly pigmented; petechial hemorrhages are present.

Intestines, Obstruction of.

I. Strangulation.

Etiology.—Constrictions by (*a*) false ligaments, (*b*) omental growths, (*c*) Meckel's diverticulum, (*d*) normal structures abnormally attached, (*e*) slits and apertures, including internal hernia. The majority of cases occur in young males.

Morbid Anatomy.—The appearance of the bowel varies with the degree of constriction. In mild cases the intestine is inflamed and the blood-vessels of the mesentery are tortuous and overfilled. In severe cases the constricted portions may undergo gangrenous change.

II. Intussusception.

Etiology.—(*a*) Early life (the majority of cases occur before the tenth year). (*b*) Excessive peristalsis.

Morbid Anatomy.—The condition is due to the slipping of a portion of the intestine into a lower segment. The tumor thus formed may be from half an inch to a foot or more in length and is sausage-shaped. At post mortem, peritonitis is usually found. The portion of the bowel affected is large and thick. There are marked swelling, congestion, and frequently œdema of the parts. The entire mass is of a deep livid-red color. The parts involved become distended with blood and hemorrhages may occur in the walls of the bowel. Gangrene is a frequent consequence, followed by sloughing of the invaginated segments, which may be discharged per rectum.

III. Twists and Knots.

Etiology.—(*a*) Male sex. (*b*) Age from thirty to forty years. (*c*) Elongated mesentery.

Morbid Anatomy.—More than half the cases occur at the sigmoid flexure; the next most common seat is the cæcum. At autopsy the sigmoid flexure is enormously distended. It may seem to occupy the whole of the abdominal cavity, not infrequently pushing the diaphragm as far as the fourth rib. The twisted bowel is congested; its color may vary from a dark red to black. The walls of the intestine are much thickened, soft, and friable, and may exhibit one or more tears.

IV. Strictures and Tumors.

Etiology.—(*a*) Congenital stricture. (*b*) Cicatricial stenosis (tubercular, syphilitic, dysenteric). (*c*) Compression and traction,—

e.g., by tumors pressing on the bowels. Organization and contraction of inflammatory exudate. (*d*) Malignant growths, such as cancer of the sigmoid flexure, descending colon, or rectum.

V. Abnormal Contents.

(*a*) Foreign bodies, such as fruit-stones, coins, pins, teeth, etc., which have usually been swallowed by children, and are generally discharged without inconvenience. (*b*) Tangled masses of round worms. (*c*) Fæces (most common). (*d*) Gall-stones. (*e*) Certain medicines, as bismuth and magnesia.

Malignant Diseases.

Morbid Anatomy.—(*a*) *Duodenum.*—Primary malignant disease must be distinguished from that of the head of the pancreas and the bile-duct. Cylindrical-celled carcinoma is the most common form. It is apt to produce annular stricture. Sarcoma is rarer, more diffuse, and occurs in nodules or plaques. The latter may lead to dilatation of the duodenum. (*b*) *Jejunum and Ileum.*—Primary disease in this portion of the bowel is rare. There is apt to be more than one growth. As elsewhere in the bowel, it is apt to lead to chronic obstruction.

Stomatitis.

Etiology.—(*a*) Extremes of life. (*b*) Most common in nursing children. (*c*) Irritant foods and poisons. (*d*) Extension of inflammation from the intestinal tract. (*e*) The infectious diseases. *Classification.*—I. Acute. (*a*) Catarrhal. (*b*) Aphthous (herpetic). (*c*) Ulcerative. (*d*) Gangrenous. (*e*) Infectious. II. Chronic.

Morbid Anatomy.—(*a*) Catarrhal. The mucous membranes are swollen, hemorrhagic, and glazy in appearance. The follicles are often very prominent. There is desquamation of epithelium and sometimes small ulcers form. The submucous tissue may be involved. (*b*) In aphthous stomatitis the tongue, the cheeks, and the tonsils are principally affected. The mucous membrane is covered with small whitish pin-point elevations, which frequently undergo necrotic changes and show more or less superficial ulcerations. The lesion always starts as a vesicle. The surrounding mucous membranes usually show evidences of acute inflammation, although they may appear normal. (*c*) In ulcerative stomatitis the points of election are the gums, the lips, and the cheek in the line of the teeth. The ulcers are gray and linear, with sharply defined, red edges. In strumous children they show a marked tendency to affect the deeper structures, sometimes causing necrosis and suppuration of portions of the jaw. In severe cases where the alveoli

are involved, the teeth may loosen and fall out. (*d*) Gangrenous stomatitis (noma) occurs between the ages of two and twelve years, is more common in girls than in boys, and follows infectious fevers (fifty per cent. of the cases occur after measles). It is characterized by the formation of a fungoid ulcer, beginning on the inner aspect of the cheek, usually with distinctly circumscribed, deeply congested edges, the surrounding tissues being markedly œdematous. The outer surface of the cheek is reddened, brawny, and indurated. The gangrenous ulcer has a decided tendency to spread, frequently involving the gums, cheek, tongue, and bones of the neighboring structures. Pneumonia not infrequently develops and is one of the causes of a fatal termination.

JAUNDICE, TOXÆMIC.

Morbid Anatomy.—(*a*) Yellowish discoloration of the skin, the mucous membranes, and the secretions and excretions, except those of the bowel, which are paler than normal. (*b*) Hemorrhages are common in the various serous membranes in the stomach, the intestines, and the subcutaneous tissues. The liver also is the site of hemorrhages of various sizes. (*c*) The liver is usually much enlarged and presents the characteristics of fatty degeneration. It is doughy to the touch, greasy on section, and of a reddish-yellow or pale-yellow color. The lobules are indistinct and show evidences of bile-staining. (*d*) The kidneys are swollen, soft, and show peripheral hemorrhages. The changes are those of acute diffuse nephritis. (*e*) The heart is flabby. The myocardium is mottled in appearance, with evidences of fatty degeneration. (*f*) The spleen is enlarged, often to twice its natural size, and full of blood. In some cases it may be small and firm.

JOINTS, DISEASES OF.

Arthritis Deformans.

Definition.—A chronic disease of the joints, characterized by changes in the cartilages and synovial membranes, with periarticular formation of bone and great deformity (Osler).

Etiology.—(*a*) Women. (*b*) Age from twenty to fifty years, although it may occur in children. (*c*) Heredity. (*d*) Infectious diseases, as gonorrhœa. (*e*) Gout and rheumatism, not common. *Classification.*—(*a*) General progressive form, which may be acute or chronic. (*b*) Heberden's nodosities. (*c*) Mono-articular form. *Seats.*—(*a*) Small joints of hands. (*b*) Knee. (*c*) Feet. (*d*) May be general.

Morbid Anatomy.—(*a*) In all forms the articular surfaces of the joints become hyperplastic and soften; absorption then takes place, the ends of the bones becoming eburnated. Dislocation may follow or the

joints may ankylose, in consequence of greatly thickened ligaments and osteophytes. The fingers are deflected to the ulnar side. In old people the disease is apt to attack the hip, knee, shoulder, or spine. There is often marked atrophy of both bones and muscles. (*b*) Heberden's nodosities are characterized by the formation of small nodes or tubercles about the dorsum of phalanges, rarely affecting large joints.

Inflammations of. (After Greene.)

Etiology.—(*a*) Traumatism. (*b*) Infectious diseases. (*c*) Rheumatism. (*d*) Gout. (*e*) Gonorrhœa. (*f*) Tuberculosis. *Classification.*—I. Acute. (*a*) Serous. (*b*) Serofibrinous. (*c*) Fibrinous. (*d*) Purulent. II. Chronic. (*a*) Serous or serofibrinous. (*b*) Gouty. (*c*) Arthritis deformans. III. Tubercular. (*a*) Miliary. (*b*) Diffuse thickening (tumor alba). (*c*) Nodular thickening (synovitis tuberosa). (*d*) Hydrops. (*e*) Empyema. IV. Syphilis. (*a*) Secondary. (*b*) Tertiary. V. Fatty degeneration and mucoid degeneration; found in old age and in cases of local defective nutrition.

Morbid Anatomy.—I. *Acute Inflammations.*—(*a*) The appearance of the inflamed synovial membranes differs in nowise from that of other inflamed serous surfaces. (*b*) The serous and serofibrinous forms usually terminate in resolution, without marked changes. (*c*) The fibrinous form frequently results in the formation of more or less extensive adhesions. (*d*) The purulent form usually leads to necrosis of the articular surfaces and often to periarticular abscesses. II. *Chronic Inflammations.*—(*a*) In chronic serous or serofibrinous inflammations the joint is distended by a thick or thin, often gelatinous secretion; the tufts are large and projecting, the synovial membrane thickened, and the joint-cartilages show degeneration as well as proliferation. Adhesions or destruction of the joint may follow. (*b*) Gouty arthritis is characterized by the deposit of the salts of uric acid in the form of granules or needle-like crystals in the articular surfaces, the capsules, ligaments, and structures surrounding the joints. Deformities arise with the formation of "tophi" (roundish chalk-like masses). The great toe and the small joints of the fingers are most frequently affected. (*c*) In arthritis deformans the articulating surfaces become hyperplastic and softened; then by attrition the ends of the bones are laid bare and become smooth and eburnated. At the edges, where friction is less, irregular nodules are formed, which are usually calcified. The ligaments and membranes become thickened and infiltrated with lime salts, causing great deformities and not infrequently ankylosis. The smaller joints of the hands and feet are usually first affected; in severe cases all the joints may be more or less involved.

There is a complete absence of uric-acid deposits. III. *Tubercular.*—(*a*) Miliary tuberculosis may be primary or secondary; it does not differ from the same affection on other serous membranes. (*b*) In diffuse thickening (tumor alba) the joint is much swollen, and on section shows tuberculous masses upon the articular surfaces or synovial membranes, together with a gelatinous exudate and cell-infiltration, or the membrane may resemble the wall of a chronic abscess, the cavity containing a puriform or turbid fluid. (*c*) In cases of nodular thickening (synovitis tuberosa) fungous masses of tuberculous matter grow from the serous membrane into the joint, which usually contains a blood-stained fluid. (*d*) Tubercular hydrops is not distinguishable from the simple form until tubercular thickening of the synovial membrane begins. (*e*) Empyema cannot be distinguished from the previous form until the fluid is drawn off. It is apt to occur in old people. IV. *Syphilis.*—(*a*) In secondary syphilis serous and serofibrinous inflammations may appear. (*b*) Tertiary syphilis may present gummata in some of the joint-structures.

Loose Bodies in the Joints.

Morbid Anatomy.—(*a*) These may be entirely free or attached by a slender stalk. (*b*) They may be composed of fibrin, (*c*) a proliferation of the synovial tufts or fatty tufts, (*d*) pieces of bone or cartilage detached by violence, or (*e*) foreign bodies which have penetrated the bone.

KIDNEY, DISEASES OF.

Amyloid (or Lardaceous) Kidney.

Etiology.—(*a*) Prolonged suppuration (tubercular or syphilitic). (*b*) Follows chronic disease of the kidney. (*c*) Follows lack of cardiac compensation in chronic Bright's disease.

Morbid Anatomy.—The amyloid kidney is usually enlarged (the condition may occasionally occur in a contracted kidney), pale in color, and firm in consistency. The capsule is adherent in places and shows petechial hemorrhages. The cortex is increased in size. The glomeruli are usually prominent, although the cortex is pale in contrast to the somewhat reddened appearance of the pyramids. The organ has a bacony or waxy appearance. If soaked in weak Lugol's solution, the amyloid material takes on a characteristic mahogany-brown color. The urine contains albumin. The tube-casts are hyaline, waxy, or finely granular. Œdema of the extremities is common.

Bright's Disease, Acute.

Definition.—An acute diffuse inflammation of the kidney.

Etiology.—(*a*) Acute infectious fevers. (*b*) Poisons,—*e.g.*, turpentine, arsenic, etc. (*c*) Traumatism. (*d*) Exposure to cold and wet.

Morbid Anatomy.—(*a*) Macroscopically the organ is swollen, tense to the touch; the capsule is stretched; the color is red and petechial hemorrhages are common on the surface. On slitting the capsule the contents of the kidney bulge out. The cortex is somewhat pale, the glomeruli appear as minute red dots. The pyramids are distinct and striated. The larger blood-vessels are overfilled and prominent. The mucous membrane of the pelvis is frequently swollen and pinkish in color. The capsule strips off easily and is somewhat thinner than normal. Microscopically the changes are those of an acute diffuse inflammation, including proliferation, desquamation, and a granular change in the cells lining the tubules. (*b*) The urine is scanty, high colored, albuminous, and contains casts and free blood. (*c*) There may be extensive œdema, with effusions into the serous cavities.

Congestion of.

Etiology.—(*a*) Traumatism. (*b*) Drugs. (*c*) Infectious fevers. (*d*) Alterations of the circulation. (*e*) Valvular lesions of the heart. (*f*) Diseases of the liver.

Morbid Anatomy.—(*a*) The kidney is large; the capsule is tense; the color is dark red. On opening the capsule the contents are soft and bulge out and the blood drips freely from the surface of the section. The dependent portions show more congestion than the cortex. (*b*) In passive congestion the organ is enlarged and firm; the capsule strips off readily; the cortex is wider than normal; the surface on section looks coarse and connective tissue is plainly visible; the cortex is of a deep-red color and the pyramids are of a purple-red.

Cystic Disease of.

Etiology.—(*a*) Congenital malformations. (*b*) Chronic nephritis.

Morbid Anatomy.—(*a*) Congenital cystic kidneys are greatly enlarged,—so much so at times as to impede labor. There may be a conglomeration of cysts varying in size from that of a pea to a small apple. In some cases no renal tissue can be seen without the aid of a microscope. The cysts are lined by a flattened epithelium and contain a fluid in which are found albumin, blood-crystals, cholesterin, triple

phosphates, and fat-drops. (*b*) In chronic nephritis small cysts are commonly found, especially about the cortex. They are usually multiple and small, but may be solitary and large.

Hydronephrosis.

Definition.—A retention of fluid in the pelvis of the kidney due to an obstruction in the ureter.

Etiology.—(*a*) Congenital deformities. (*b*) Twists of the ureter. (*c*) Calculi. (*d*) Morbid growths. (*e*) Cicatricial bands.

Morbid Anatomy.—There is an accumulation of non-purulent fluid in the pelvis of the kidney, which by steady pressure produces an atrophy of the organ and a gradual distention of its pelvis. In extreme cases the kidney may be converted into a large cyst, with some imperfect septa. There may be an enormous quantity of the contained fluid or only a few ounces. It is yellowish in color and contains urea, uric acid, and sometimes albumin and sugar. There is usually compensatory hypertrophy of the opposite kidney.

Interstitial Nephritis.

Morbid Anatomy.—The kidney is contracted and indurated, the cortex diminished, the capsule adherent, the surface rough, and numerous cysts are seen. The process is chronic and often associated with arteriosclerosis.

Movable Kidney.

Etiology.—(*a*) Female sex. (*b*) Absorption of perirenal fat. (*c*) Repeated pregnancies. (*d*) Traumatism. (*e*) Displacement by tumors.

Morbid Anatomy.—As a rule, the displacement is not great. The kidney usually moves downward or upward and inward, generally rotating so that the outer border and upper end move forward and the hilum is directed inward and backward. Nearly all cases are associated with a medial displacement of the colon. The right kidney is the one most frequently affected.

Perinephritic Abscess.

Etiology.—(*a*) Traumatism. (*b*) Extension of inflammation from the kidney or from neighboring organs. (*c*) Perforation of the bowel. (*d*) Infectious fevers, especially in children.

Morbid Anatomy.—The kidney is surrounded by pus, especially posteriorly. The abscess-cavity is usually extensive. The pus is often offensive and may have a distinctly fecal odor. It may burrow and discharge into the lung, bowel, peritoneum, or bladder. It may follow the psoas muscle and appear in the groin.

Pyelitis.

Definition.—Inflammation of the pelvis of the kidney.

Etiology.—(*a*) Tuberculosis. (*b*) Infectious fevers. (*c*) Calculi. (*d*) Cystitis. (*e*) Tumors. (*f*) Drugs. (*g*) Cold and wet. *Classification.*—(*a*) Simple catarrhal. (*b*) Purulent. (*c*) Calculous.

Morbid Anatomy.—(*a*) In simple acute pyelitis the mucous membrane of the pelvis is swollen, hemorrhagic, and turbid. (*b*) In the purulent form the mucous membrane is swollen and covered with a cream-like exudate of a yellowish or yellowish-green color. Ecchymoses are common. The kidney itself is enlarged, softened, œdematous, grayish in color, and shows little distinction between cortex and medulla. Areas of necrosis or miliary abscesses are distributed through the kidney substance. The kidney may attain the size of a human head. It is usually firmly adherent to the adjacent organs, tissues, and vessels. A quart or more of pus may be contained in the cavity; in these extreme cases all appearance of the gland substance may be lost. (*c*) In calculous pyelitis the mucosa is roughened, grayish in color, and thickened. There are also more or less dilatation of the calyces and flattening of the papillæ. (*d*) Following this condition may be pyelonephritis, atrophy of the kidney substance, and finally pyonephrosis. (*e*) After the renal substance has been destroyed, if the pelvic orifice is still obstructed, the pus may become inspissated and ultimately impregnated with lime salts.

Tumors of.

1. Benign.—(*a*) Fibromata are most common. (*b*) Lipomata. (*c*) Angiomata. (*d*) Lymphadenomata (or lymphomata).

2. Malignant.—(*a*) Cancer may be primary or secondary; it is comparatively rare and of the encephaloid variety. (*b*) Sarcoma may be primary or secondary. It is more common than cancer, usually occurs in children, and may attain to an enormous size.

LARYNGITIS, ŒDEMATOUS.

Etiology.—(*a*) Septic infection. (*b*) Traumatism. (*c*) Certain drugs. (*d*) Chronic visceral diseases,—*e.g.,* Bright's disease. *Classification.*—(*a*) Inflammatory, which may be septic or non-septic. (*b*) Non-inflammatory or dropsical.

Morbid Anatomy.—(*a*) The aryepiglottic folds and the ventricular bands are the parts chiefly affected. The vocal cords are seldom included, but the œdema may go below them. (*b*) The exudation may be serous, seropurulent, or purulent, and may or may not be blood-stained. (*c*) In very severe cases the larynx may be entirely closed.

LARYNGITIS, SYPHILITIC.

Classification.—(*a*) Congenital. (*b*) Acquired, which may be secondary or tertiary.

Morbid Anatomy.—(*a*) In the secondary form there is erythema, with symmetrical, superficial, whitish ulcers on the cords or ventricular bands. (*b*) Mucous patches are occasionally seen. (*c*) In the tertiary form true gummata may appear towards the base of the epiglottis. These break down, producing deep flask-shaped ulcerations, which may heal by connective tissue that shrinks and produces stenosis. (*d*) Islands of connective tissue commonly appear between the cicatrices and form inflammatory excrescences. (*e*) The neighboring cartilages may show necrotic changes. (*f*) A fatal termination may result from perforation of an artery.

LEPROSY.

Definition.—An infectious disease characterized by the formation of a node or nodule, and due to the leprosy bacillus.

Etiology.—The *Bacillus lepræ* has many points of resemblance to the bacillus of tuberculosis. It, however, stains more readily, is more easily decolorized, and is present in far greater numbers in the lesions which it causes. *Classification.*—(*a*) Tubercular. (*b*) Anæsthetic.

Morbid Anatomy.—(*a*) The tubercular form starts as a small red spot, which either disappears or gives rise to the formation of inflammatory nodules of a brownish-red color, somewhat soft in consistency, and is said to resemble a strawberry. The primary lesion is found most frequently in the skin of the face, in the surfaces of the knees, of the elbows, of the hands, and of the feet. It may also involve the conjunctiva and the mucous membrane, cornea, and the larynx. This form of the disease is apt to be exceedingly chronic, the surrounding tissues showing marked fibroid changes. The tubercles rarely break down. (*b*) In the anæsthetic form the leprous process gradually involves the peripheral nerves, obliterating them and causing marked trophic changes, consisting in necrosis and ulceration with extensive loss of substance, as of fingers, toes, and even limbs. There is great loss of hair and the face often shows marked ravages of the disease. Death results not infrequently from laryngeal complication or aspiration pneumonia.

LEUCOCYTHÆMIA (OR LEUKÆMIA).

Definition.—A form of primary anæmia characterized by great increase of the white corpuscles and by marked structural changes in the lymphatic glands.

Etiology.—Not definitely known; usually ascribed to changes in the blood-making organs. *Classification.*—(*a*) Splenic. (*b*) Medullary. (*c*) Lymphatic. (*d*) Combinations of the three forms.

Morbid Anatomy.—As a rule, the patient is apparently well preserved, but in some cases emaciation may be extreme. The skin has a peculiar lemon-yellow color. The mucous membranes are blanched. The amount of adipose tissue is frequently increased and of a peculiar punctate appearance, owing to the presence of petechial hemorrhages. The blood is pale in color,—often milk-white. It rarely clots with any rapidity. The organs in general are pale; the liver, spleen, and lymphatic glands are usually markedly enlarged. The heart is pale, flabby, and frequently fatty in appearance. (*a*) In splenic anæmia, which is a comparatively rare form of the disease, the spleen is markedly enlarged, somewhat firm in consistency, and of a reddish-brown color. The Malpighian bodies are frequently obliterated, their place often being taken by grayish-white, circumscribed tumors throughout the organ. The hyperæmia in some cases is excessive, and rupture of the spleen is said to have occurred from this cause. Dropsy from pressure on the abdominal viscera may result. As in other forms of leukæmia, the bone-marrow may show decided changes, especially in the long bones. Instead of fatty tissue there may be splenization, or it may resemble the consistent matter which forms the core of an abscess. Microscopical examination of the blood shows that the increased white corpuscles are largely myelocytes. (*b*) Medullary leukæmia very seldom occurs as an inflammatory process. Where the marrow changes are excessive, the flat bones—as, *e.g.,* the sternum—undergo alterations similar to those occurring in the long bones. (*c*) In lymphatic leukæmia the lymphatic glands throughout the body, especially those of the neck, the axillary and inguinal regions, and the glands of the mesentery and the intestines, show marked involvement. The liver, as well as the spleen, is enlarged and may exhibit marked structural changes. The lymphatic glands in general are swollen, pale in color, firm to the touch, and seldom suppurate or show any tendency to run together. The spleen, liver, and other lymphatic glands often show marked thickening of their capsules. On section the glands are somewhat resistant, and often exhibit nodule-like bodies, which are firm in consistence and largely composed of proliferating leucocytes and connective tissue. Microscopical examination of the blood shows that the marked increase of the leucocytes is in the lymphocytes. (*d*) The blood in leukæmia in general shows somewhat marked diminution of the amount of hæmoglobin; the red corpuscles are reduced in number, sometimes markedly so. The white corpuscles are enormously

and permanently increased, so that one white to twenty red, or even one to one, is not uncommonly found. The characteristic feature of leukæmic blood is the alteration of the relative proportions of the various white corpuscles the one to the other.

LIVER, DISEASES OF.

Acute Yellow Atrophy.

Definition.—An acute disease of the liver, presumably of infectious origin, characterized by a rapid fatty degeneration of the organ, with invariably fatal termination.

Etiology.—(*a*) A septic micro-organism(?). (*b*) The ordinary micro-organisms of suppuration and infectious diseases have been found in this condition. (*c*) Certain poisons,—*e.g.*, phosphorus. (*d*) Female sex. (*e*) Pregnancy or the puerperium.

Morbid Anatomy.—The liver is greatly reduced in size,—one-half to one-third; in one of my cases, however, the condition had been preceded by hypertrophic cirrhosis, and the organ weighed over five pounds. The liver is thin, flattened, and flabby, the capsule is wrinkled, and the gland is of a pale-yellow color. Both on the surface and on section may be seen a number of orange-yellow patches, in the centre of which are usually marked hemorrhagic areas. The remainder of the liver is of a yellowish-brown or mottled color. The outlines of the lobules are very indistinct. The bile-ducts and gall-bladder are empty. The other organs are usually stained with bile and present numerous hemorrhages, especially on the surface. The spleen is enlarged and the heart and kidney show marked granular change. The color of the liver in acute yellow atrophy depends on the time at which death took place: in the earlier stages the organ is ochre-yellow, in the later stages it is mottled, and if much blood be present it is grayish red.

Amyloid.

Etiology.—(*a*) Prolonged suppuration, tubercular or syphilitic. (*b*) Infectious fevers. (*c*) Chronic visceral diseases with cachexia.

Morbid Anatomy.—The liver is large in size, smooth in outline, and pale in color. The edges are distinctly rounded; small hemorrhages are common on the surface. On section the surface is anæmic, semi-transparent, and infiltrated. It presents the characteristic lardaceous or waxy appearance. The process may be a localized or a generalized one; in either case staining by Lugol's solution is never uniform, as the diseased brown spots appear only in certain areas. The characteristic coloration may be seen upon the lining of both hepatic and portal vessels.

Cancer of.

Etiology.—(*a*) Male sex. (*b*) Age, from forty to sixty years. (*c*) Primary cancer elsewhere. (*d*) Heredity.

Morbid Anatomy.—I. *Secondary Cancer.*—Most common. Histologically shows same structure as primary growth, which is usually in the stomach, bowel, or pancreas. The liver is enormously enlarged, irregular, and nodular. The nodules are usually symmetrical, often superficial, flattened, discrete, and umbilicated; they may be more or less evenly distributed throughout the liver. On section whitish masses of varying size are seen, contrasting with the red color of hepatic tissue, the yellow staining of bile, pigmentation due to blood, and the light-yellow areas of fatty degeneration. The cancerous masses may undergo fatty degeneration, suppuration, or fibroid change. II. *Primary Cancer.*—Rare. (*a*) Massive. Causes great enlargement. On section the mass is uniform grayish white in color, somewhat firm, and distinctly outlined from the liver substance. (*b*) Nodular. Large and small nodules are scattered throughout the organ. These usually consist of a primary growth and numerous secondary nodules. (*c*) Cancer with cirrhosis is rare. Liver not much enlarged. Surface of section is grayish yellow, studded with nodular yellowish masses.

Cholecystitis, Acute Infectious.

Definition.—An inflammation of the gall-bladder due to the introduction of pyogenic micro-organisms.

Etiology.—(*a*) Micro-organisms,—for example, the *Bacillus coli communis* and the typhoid bacillus, pneumococcus, staphylococcus, and streptococcus. (*b*) Gall-stones. (*c*) Extension of inflammation from the bile-ducts.

Morbid Anatomy.—The gall-bladder is distended, the walls are thickened and tense. The mucous membrane is swollen, hyperæmic, and may be covered with a purulent exudate. The contents of the bladder are dark in color, and may be mucopurulent or hemorrhagic. Gall-stones are frequently present. The cystic duct is often obliterated. There may be adhesions with the bile-duct or omentum.

Cholelithiasis.

Definition.—The formation of gall-stones within the gall-bladder or in the ducts leading to or from it.

Etiology.—(*a*) Female sex. (*b*) Age, fifty per cent. over forty years. (*c*) Sedentary habits. (*d*) Overeating.

Morbid Anatomy.—(*a*) The calculi are usually multiple; they

may be single. They vary in size as well as in number. When multiple they are faceted, sometimes mulberry-shaped. They are of a dark bluish or greenish color. On section there is a nucleus consisting of epithelium, rarely a foreign body, then comes a layer of inspissated bile-salts, the outer covering being cholesterin. There may also be bile-acids, fatty acids, salts of calcium and magnesium, with a trace of iron and copper. These stones are usually firm in consistence or they may be friable. (*b*) The gall-stones may lead to impaction of the gall-bladder or to obstruction of the cystic and common ducts or even of the bile-duct alone. There may be formation of a fistula, external or internal, with escape of bile. The bladder itself is much thickened, sometimes dilated, sometimes smaller than normal.

Cirrhosis of.

Definition.—Under this heading are classified various forms of disease of the liver characterized by a marked increase of its connective tissue, which may be capsular, interlobular, or intralobular, with or without increase or decrease in the size of the organ.

Etiology.—(*a*) Alcohol. (*b*) Certain infectious diseases,—*e.g.*, syphilis, tuberculosis, malaria, scarlet fever. (*c*) Micro-organismal infection. (*d*) Mechanical obstruction to the onward flow of the blood. (*e*) Rickets. (*f*) Anthracosis. *Classification.*—(*a*) Alcoholic. (*b*) Fatty. (*c*) Hypertrophic. (*d*) Capsular. (*e*) Syphilitic. (*f*) Cyanotic. (*g*) Malarial. (*h*) Scarlatinal. (*i*) Tubercular. (*j*) Rhachitic. (*k*) Anthracotic.

Morbid Anatomy.—(*a*) In the atrophic cirrhosis of Laennec the organ is greatly reduced in size, and later altered in shape. The surface is irregular and nodular. The nodules are usually small, although in some cases they may be greatly increased in size. The tissue is firm, hard, and resistant to the knife. The surface of section presents a mottled appearance, the lobules being divided by bands of connective tissue. The liver substance itself is of a yellowish or greenish-yellow color. The areas of connective tissue are gray. (*b*) In fatty cirrhosis the organ is enlarged, somewhat smooth, although often slightly granular. It is paler than normal and of a yellowish-white color. It is firm and resistant to the knife. The capsule is opaque and often much thickened. The peritoneal cavity usually contains ascitic fluid. The membrane is opaque and thickened. Chronic involvement of the stomach and small intestine is always present. The spleen is enlarged; the kidneys are often cirrhotic. Owing to interference with the portal circulation by the cirrhotic liver, extensive compensatory circulation is formed. The abdominal vessels above and below the umbilicus are markedly

enlarged. Around the umbilicus is found the caput of Medusa. Acute tuberculosis of the peritoneal cavity is not infrequently associated. (*c*) Hypertrophic cirrhosis is most common in young men. The organ is enlarged, but the outline is normal. The surface is usually smooth and the color of an olive-green; the consistency of the organ is increased and the capsule is thickened. The surface of section is uniformly greenish yellow and the lobules may be separated by distinct bands of connective tissue. The spleen is greatly enlarged. Jaundice is a marked symptom of this disease. Ascites is usually absent. (*d*) In capsular cirrhosis there is enormous thickening of the capsule, which is irregular and somewhat wrinkled, producing great contraction of the liver. The organ itself is rarely markedly cirrhotic, its tissue being usually soft. Chronic capsulitis of the spleen, chronic perisplenitis and ascites are often present. The kidneys usually show granular change. (*e*) In syphilitic cirrhosis the liver is markedly irregular in shape, being divided into peculiarly shaped lobes by extensive bands of fibrous tissue traversing the organ in indefinite directions. In one of my cases over forty distinct lobulations were present. The cut surface is mottled, often fatty in appearance, and shows the presence of gummata or of syphilitic scars. The connective-tissue bands are of a gray or reddish-gray color. (*f*) For cyanotic cirrhosis see Passive Congestion of the Liver. (*g*) In malarial cirrhosis the liver is markedly enlarged, commonly extending to the level of the umbilicus. It is firm in consistence, of a dark-red color, smooth in outline, and bleeds freely on section. (*h*) Klein has pointed out that chronic interstitial hepatitis may follow an attack of scarlet fever, which may account for some cases of cirrhosis of the liver in children. (*i*) For tubercular cirrhosis see Tuberculosis. (*j*) Rhachitic cirrhosis is a form of the disease in which there is a marked increase of connective tissue around the individual lobules. (*k*) Anthracotic cirrhosis occurs in coal-miners, in whom the coal-dust may occasionally reach the liver in sufficient quantities to cause a marked connective-tissue formation about the portal canal. (Welch.)

Congestion of.

Etiology.—(*a*) Acute infectious diseases. (*b*) Traumatism. (*c*) Extension of inflammation,—*e.g.*, from the intestines. (*d*) Valvular heart-disease. (*e*) Pressure of tumors. (*f*) Other mechanical obstructions to the circulation. *Classification.*—(*a*) Active. (*b*) Passive.

Morbid Anatomy.—(*a*) The post-mortem appearances of active congestion are not characteristic. The liver is swollen, dark in color,

and full of blood; the hyperæmia is not limited to any one portion of the liver substance. (*b*) In passive congestion the liver is large in size, smooth or slightly granular in outline, and of a distinctly mottled appearance. The surface of section presents the characteristic nutmeg appearance, due to a marked congestion occurring in the central veins, the congested area being of a reddish-brown color. This is surrounded by a large area of a pale-yellowish color, with a third zone of cellular infiltration and new connective tissue. In rare cases this order is reversed, the congested area occurring at the periphery of the lobe and the lighter or fatty areas towards the centre. In chronic and well-marked cases there may be considerable induration with shrinkage of the liver substance so that the hypertrophy gives place to an atrophy.

Fatty.

Etiology.—(*a*) Middle life. (*b*) Alcohol. (*c*) Sedentary habits. (*d*) Infectious fevers. (*e*) Certain poisons. (*f*) Interference with local or general circulation. *Classification.*—(*a*) Fatty degeneration. (*b*) Fatty infiltration.

Morbid Anatomy.—(*a*) The liver may be increased or diminished in size. The capsule may be smooth or wrinkled. The consistence is usually somewhat decreased; the organ is paler than normal and somewhat mottled in appearance. The surface of section is smooth, usually bloodless, and imparts a greasy stain to the knife. The general color is a dull gray or grayish yellow. (*b*) In fatty infiltration the liver is often markedly enlarged, normal in outline, smooth to the touch, and of a somewhat pale, excessively fatty color. Globules of fat may be readily expressed with a knife.

Hepatitis, Suppurative.

Definition.—Abscess of the liver.

Etiology.—(*a*) Traumatism. (*b*) Extension from neighboring organs,—*e.g.*, the bowel and the pleura. (*c*) Pyæmia. (*d*) Amœbic dysentery. *Classification.*—(*a*) Pyæmic hepatitis. (*b*) Portal pyæmia. (*c*) Pyosepticæmia or multiple abscess. (*d*) Tropical or endemic hepatitis. (*e*) Suppurative cholangeitis.

Morbid Anatomy.—(*a*) In multiple abscess the change in the liver depends upon the number of the abscesses. If these be few, the liver walls may be comparatively little altered; if they are very many, the liver is apt to be enlarged, softened, and friable. The abscesses themselves appear as minute foci which are non-encapsulated, the centre containing a thick white, yellow, or greenish pus surrounded by a zone of congestion. The abscesses may number from five to ten, or

many hundreds. These multiple abscesses frequently arise from pyæmic embolism of the portal vein or hepatic artery or vein, or they may result from a cholangeitis. They may be generally distributed or appear in clusters. (*b*) Large abscesses occur in two forms,—as the large chronic encapsulated abscess surrounded by a pyogenic membrane or as the tropical or amœbic abscess. (See Dysentery.) The large abscess is usually single; there may be two or more. The right lobe is usually affected. There is a distinct limiting membrane. The pus is usually of a greenish-yellow color and often of a disagreeable odor. The surrounding substances often show but few changes, except as the result of pressure.

Sarcoma of.

This may be primary or secondary. The most frequent variety is the secondary melano-sarcoma following sarcoma of the eye, of the skin, or of the penis. In these cases the liver is greatly enlarged, weighing as much as fifteen pounds, and the secondary nodules, which are of a black or slate color, are usually uniformly distributed throughout the liver. In primary sarcoma of the liver there are but few nodules, and these reach a large size, measuring at times five or six inches in diameter. Metastases to other organs often occur, though other portions of the liver may escape.

LUNGS, DISEASES OF.

Abscess of.

Etiology.—(*a*) Pyogenic micro-organisms. (*b*) Pneumococcus. (*c*) Tubercle-bacillus. (*d*) Gonococcus. (*e*) Septic infection from distant foci. *Classification.*—(*a*) Single or large abscess. (*b*) Minute or multiple abscess.

Morbid Anatomy.—Solitary abscesses of the lung are comparatively rare. They are usually the result of extension from neighboring parts, as the pleura, liver, or mediastinum. The abscess is encapsulated and contains a greenish-yellow pus, which often has an offensive odor. (*b*) Multiple abscesses are common in pyæmia. They are usually superficial, often wedge-shaped, and rarely encapsulated. They are at first firm, grayish-red in color, and surrounded by a zone of hyperæmia. Later they become distinctly purulent. The pleura is usually covered with a greenish lymph. Perforation of the pleura, causing empyæma, may occur.

Bronchopneumonia—see Pneumonia, Catarrhal.

Emphysema of.

Definition.—Dilatation of the air-vesicles due to some weakening of the lung structure and a dilating force, usually expiration.

Etiology.—(*a*) Chronic cough. (*b*) Certain occupations, as glass-blowing. (*c*) Senile changes. (*d*) Traumatism. *Classification.*—I. Acute. (*a*) Simple or mechanical. (*b*) Interstitial. II. Chronic. (*a*) Substantial or hypertrophic. (*b*) Senile or atrophic.

Morbid Anatomy.—On inspection the thorax is increased in its anteroposterior diameter and barrel-shaped. The clavicles are prominent, as are also the sternum and the costal cartilages. The interstitial spaces are enlarged and the sternal fossa is deep. The neck appears to be shortened. Dilated veins may be seen along the line of the attachment of the diaphragm. The back is rounded and the curve of the spine increased. On removing the sternum the anterior mediastinum is found to be completely occupied by pulmonary tissue and the pericardial sac may be entirely covered by the lungs. The latter are large, light in color, and deeply pigmented in places. They are inelastic and do not collapse, but pit readily on pressure. The edges are distinctly rounded and obtuse. Beneath the pleura, especially about the anterior margins and at the inner surface of the lobe near the right, enlarged air-vesicles of bladder-like appearance may be seen, varying in size from that of a pea to a hen's egg. The mucous membrane of the large bronchi may be rough and thickened; bronchiectasis may also be present. The right heart is dilated and hypertrophied; the pulmonary artery may be enlarged and show atheromatous changes.

Gangrene of.

Etiology.—(*a*) Follows inflammation of the lungs. (*b*) Saprophytic micro-organisms.

Morbid Anatomy.—(*a*) The lower lobe is usually affected, the peripheral portions rather than the central. The gangrenous part is larger, heavier, and of a greenish-black color. The outer tissues are intensely œdematous, next is an area of deep congestion, and then a cavity with shreddy irregular walls containing an offensive greenish fluid. The pleura may be simply inflamed and contain an abnormal amount of exudate, or it may be perforated, causing a pyopneumothorax. The gangrenous material gives rise to an intense bronchitis, the bronchial tubes containing a thin, highly offensive pus. (*b*) Embolic processes are common, abscesses of the various organs, especially the brain, being the result.

New Growths in.

Classification.—(*a*) Benign, such as fibroma, adenoma, and chondroma. (*b*) Malignant growths, which may be either primary or secondary, carcinoma or sarcoma.

Morbid Anatomy.—Primary growths in the lung are rare. Secondary sarcoma and secondary carcinoma are comparatively common. In primary growths one lung only is involved; in secondary growths, both lungs. Secondary cancer is more frequent in women than in men. These secondary growths may be scirrhous, encephaloid, epitheliomatous, or colloid cancer, or melanosarcoma. In malignant diseases of the lungs pleurisy, generally of a hemorrhagic type, is commonly present. The tracheal or bronchial glands are usually the seat of metastatic growths. The condition is most common in middle life.

Passive Congestion of.

Etiology.—(*a*) A mechanical obstruction to the circulation. (*b*) Chronic disease requiring the recumbent position. (*c*) Disease of the central nervous system,—*e.g.,* cerebral apoplexy.

Morbid Anatomy.—(*a*) In mechanical congestion there is obstruction to the return of the blood to the heart. The lungs are voluminous, russet-brown in color, and cut and tear with difficulty. On section they are of a maroon tinge, which soon gives way to a vivid red on exposure to the air. (*b*) In hypostatic congestion the bases of the lungs are deeply cyanosed, the posterior parts particularly are engorged with blood and serum, and in some instances portions of the tissue will sink in water. In prolonged coma the hypostatic congestion may be associated with patches of consolidation due to the aspiration of food into the air-passages. (*c*) In cerebral apoplexy the bases of the lungs are deeply engorged and heavy and on section exude a bloody serum. This congestion is most marked in and may be confined to the hemorrhagic side.

Pleurisy.

Etiology.—(*a*) Exposure to cold and wet. (*b*) Traumatism. (*c*) Extension of inflammation from neighboring organs. (*d*) Pyogenic micro-organisms. (*e*) Infectious fevers. (*f*) Infectious granulomata. (*g*) Malignant tumors. *Classification.*—I. Acute. (*a*) Serous. (*b*) Serofibrinous. (*c*) Fibrinous. (*d*) Purulent. (*e*) Hemorrhagic. II. Chronic. (*a*) Pleurisy with effusion. (*b*) Dry pleurisy. (*c*) Primitive dry pleurisy.

Morbid Anatomy.—I. (*a*) In acute pleurisy the serous, serofibrinous, and fibrinous forms differ in the character of the exudate. In all

three the serous membrane is at first red, sticky, and lustreless; later it becomes somewhat pale, thickened, and lustreless. The pleural cavity may contain a liquid inflammatory exudate varying in amount from a few cubic centimetres to one or more litres. Its specific gravity is above 1017; it is rich in fibrin and contains many leucocytes and some red corpuscles and swollen endothelial cells. The sero-fibrinous exudate contains more fibrin and less fluid. The characteristic of the fibrinous exudate is the so-called bread-and-butter appearance. The fibrinous deposit varies in thickness from a millimetre to a centimetre or more. (*b*) Purulent pleurisy may follow the other acute forms or may be primary. The serous membranes are covered with a creamy exudate and the pleural cavity contains from a few cubic centimetres to a litre or more of pus. This is of a greenish-yellow color, often has an offensive odor, and is frequently associated with tuberculosis of the membranes. (*c*) Hemorrhagic pleurisy may be due to asthenic conditions—tuberculosis, cancer—or may occur in perfectly healthy individuals. In the latter case it must be remembered that during aspiration the lung may be wounded and any fluid present thus become mixed with blood. The pleural cavity contains blood, which is usually in a fluid condition varying considerably in density. The serous membranes are generally inflamed and stained with blood-coloring matter. II. (*a*) In chronic pleurisy with effusion the condition may persist for months without undergoing alteration. The appearances are nearly those of an acute pleurisy. (*b*) Chronic dry pleurisy is the result of an acute pleurisy in which the exudate is partially absorbed and the material remaining undergoes organization. This occurs usually at the base and may cause marked flattening of the chest. Small pockets of fluid are often found. The lung itself is compressed, airless, and fibroid. It is frequently impossible to separate the layers of pleura. (*c*) Primitive dry pleurisy may be limited in extent or universal. The layers of pleura are firmly adherent to one another and, especially about the lower lobe, are much thickened. In cases of tubercular origin they present firm fibroid masses and small tubercles, and between the layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. The condition may be unilateral or bilateral, and may be accompanied by a similar condition in the pericardium and peritoneum. The bronchi may present marked dilatations and the lung tissue is more or less sclerosed. In diaphragmatic, encysted, and interlobar pleurisy the morbid anatomy does not differ essentially from that already described.

Pneumonia, Catarrhal (Bronchopneumonia).

Definition.—An acute inflammation of the lungs, involving both bronchial tubes and air-vesicles, due to aspiration of irritants.

Etiology.—(*a*) Micro-organisms,—*e.g., Bacillus lanceolatus, Staphylococci, Streptococci,* the diphtheria bacillus, and the bacillus of pneumonia. (*b*) Inhalation of irritant gases and vapors. (*c*) Infectious fevers. (*d*) Extension of inflammation from neighboring parts.

Morbid Anatomy.—Macroscopically the lung is larger, heavier, and firmer to the touch. On section the surface is somewhat dark red in color, but distinctly mottled, and may drip blood. On palpation nodular bodies can be felt, surrounded by crepitant areas. The nodules may resemble gray hepatization; typical ones contain a central bronchiole which is filled with tenacious purulent mucus and surrounded by a grayish-red elevated area of consolidation. Minute hemorrhages are common in the lung and on the pleural surfaces. Emphysema is seen on the anterior and upper portions of the lung, especially within the inflamed areas. Fibroid changes seldom follow bronchopneumonia.

Pneumonia, Chronic Interstitial.

Etiology.—(*a*) Various acute inflammations (rare). (*b*) Tuberculosis. (*c*) Chronic pleurisy. (*d*) Chronic poisoning. (*e*) Syphilis.

Morbid Anatomy.—The disease is usually unilateral; the chest on the affected side is sunken, deformed, and the shoulder depressed. The opposite side is usually emphysematous. On opening the chest the affected lung is seen to be airless, firm, hard, and very resistant to the knife. On section grayish fibroid tissue of variable amount, through which pass the blood-vessels and bronchi, is found. The latter may be more or less dilated. The unaffected lung is emphysematous and occupies the greater portion of the mediastinum. The heart is drawn over to the affected side. It is hypertrophied and there may be atheromatous changes in the pulmonary artery. Amyloid changes in the other viscera may be found.

Pneumonia, Lobar (Croupous or Fibrinous Pneumonia).

Etiology.—(*a*) Adult life. (*b*) Male sex. (*c*) Infectious diseases. (*d*) Alcoholism. (*e*) Exposure to cold and wet. (*f*) The *Diplococcus pneumoniæ* or *Micrococcus lanceolatus* of Fränkel is present in a large proportion of cases. It occurs in pairs, surrounded by a lanceolate capsule. It is readily demonstrated in cover-glass preparations stained by Gram's method. It is found in the bronchial secretions and in sections of the affected lung.

Morbid Anatomy.—The process is divided into three distinct stages,—hyperæmia, red hepatization, and gray hepatization. (*a*) In the stage of engorgement the lung is heavier, more solid, firmer, and redder than normal. The surface of section exudes fluid and serum. The lung still crepitates, though not as distinctly as the healthy tissue. The excised portions partially float. Microscopically the capillaries in the affected area are overfilled, tortuous, elongated, and push themselves into the alveolar spaces. The air-cells are filled with bloody exudate containing red and white blood-cells and proliferated epithelium. The alveolar epithelium is swollen and in some places desquamated. (*b*) In cases of red hepatization the lung is still larger, heavier, firmer, and of a deep-red color. It is airless, does not collapse on exposure to the atmosphere, and excised portions sink in water. The surface of the lung is covered with a more or less extensive layer of fibrin, which forms a false membrane that contrasts markedly with the smooth shiny appearance of the unaffected portions of the lung. The surface may retain the impression of the ribs. On section the lung is dry and reddish brown. It is exceedingly friable. Careful inspection shows that the surface is distinctly granular, the granules being due to fibrinous plugs, which can be scraped off with a knife together with a reddish viscid serum. The smaller bronchi often contain fibrinous clots, which may also be found filling the blood-vessels at the root of the lung. (*c*) In gray hepatization the color varies from a reddish brown to a grayish white. The surface is more moist, the exudate more turbid. The granules are less distinct and the lung-tissue is still more friable. The exudate is softened. The cell-elements are disintegrated and prepared for absorption. In advanced stages it may be called a purulent infiltration. It is probable that in this stage resolution could not take place. Abscesses of varying sizes may develop. *Other Lesions in Croupous Pneumonia.*—(*a*) Usually the right lung is affected, the process being confined to the lower lobe. The unaffected portions are congested and œdematous. (*b*) At the time of death the bronchi contain, as a rule, a mucous or mucoserous secretion tinged with blood, more rarely the tenacious mucus so characteristic of pneumonic sputum. The mucous membrane is reddened, but not usually swollen. The affected areas and small bronchi contain fibrinous plugs, which may extend into the larger tubes and form perfect casts. The bronchial glands are swollen, soft, and hemorrhagic. (*c*) The pleural surface over the inflamed area shows a more or less extensive exudate, which may be serous or fibrinous. *Lesions in Other Organs.*—(*a*) The distention of the right heart is marked. The cavities are often distended with firm tenacious coagula. (*b*) The spleen is often enlarged. (*c*) The

kidneys show cloudy swelling and often acute parenchymatous changes. (*d*) Pericarditis is not infrequent; it is usually associated with pneumonia of the left side or with double pneumonia. (*e*) Endocarditis is more common. It is often of a malignant type. (*f*) Meningitis, usually cortical, is not infrequent and is apt to be associated with malignant endocarditis. (*g*) The liver shows parenchymatous changes and often extreme engorgement of the hepatic veins. *Complications.*—(*a*) Pleurisy. (*b*) Empyema. (*c*) Pericarditis, most common in children. (*d*) Endocarditis is more frequent than pericarditis. (*e*) Myocarditis is rare. (*f*) Meningitis is perhaps the most serious complication. (*g*) Otitis media is not unusual in children. (*h*) Abscesses or gangrene in the lung occasionally occur. (*i*) Severe and often fatal toxæmia may develop with a comparatively slight lesion in the lung. *Terminations.*—(*a*) Liquefaction and resolution. (*b*) Suppuration. (*c*) Abscesses or gangrene. (*d*) Fibroid changes or carnification.

Pneumonoconiosis.

Definition.—A fibroid condition of the lung, often associated with tuberculosis, produced by the introduction of particles of mineral or metallic substances. Various names have been applied, depending upon the nature of the inspired dust,—*e.g.*, anthracosis, siderosis, calcicosis, etc.

Etiology.—(*a*) Coal-mining. (*b*) Steel-grinding. (*c*) Stone-cutting, etc.

Morbid Anatomy.—The affected lungs are harder, firmer, and frequently smaller than normal. They are usually of a blue-black or a yellowish or buff color. Even where the inspired dust is pale, the lungs are apt to be of a deep color. In advanced forms of anthracosis an ink-like juice may exude from the cut surface. Oxide of iron produces a reddish color of the lung. On section condensed portions of highly fibroid lung are seen. The surface of section commonly exhibits numerous raised points, which give it a coarse granular appearance. These raised points are found to be small, thickened, fibroid bronchial tubes protruding above the surface. Chronic bronchitis is invariably present and in long-standing cases emphysema occurs. The bronchial and peribronchial glands as well as the peribronchial lymph-nodules are frequently the site of intense pigmentation. Occasionally the pigmentation may be found also in the liver and spleen.

Pneumopericardium.

Air in the pericardial sac. It is nearly always caused by perforation from without, as in cases of stab-wounds, though it may be due to per-

foration from the lungs, œsophagus, or stomach. Its pathology resembles that of pneumothorax.

Pneumothorax.

Etiology.—(*a*) Traumatism. (*b*) Tuberculosis of the lung rupturing into the pleura. (*c*) Other infectious granulomata. (*d*) Malignant growths.

Morbid Anatomy.—The thorax is usually distended; the intercostal spaces may be obliterated. The introduction of a trocar allows the escape of air. The pericardium and heart are pushed or drawn to the opposite side. The serous membranes are inflamed and a serous or purulent fluid is present. The lung is usually compressed and carnified and may be adherent to the chest-wall at the apex. It is frequently the site of caseous nodules or cavities at the apex.

MALARIA.

Etiology.—A true hæmatozoon. Three varieties have been described. (*a*) Tertian. (*b*) Quartan. (*c*) Æstivo-autumnal. *Classification.*—(*a*) Acute malarial fever, which may be quotidian, tertian, or quartan. (*b*) Pernicious malaria. (*c*) Chronic malarial cachexia.

Morbid Anatomy.—(*a*) Cases of simple malarial fever are rarely fatal. The blood shows disintegration of red corpuscles and an accumulation of pigment is thereby formed. The spleen is enlarged, dark in color, and may show pigmentary deposits. (*b*) In pernicious malaria the blood contains enormous numbers of the parasites. The red corpuscles are in all stages of destruction and the serum is tinged with hæmoglobin. The spleen is moderately enlarged. The pulp is soft, chocolate-colored, and turbid; it contains large numbers of red corpuscles and parasites and the amount of pigment is greatly increased. The liver is swollen and presents areas of focal necrosis and capillary thrombosis. Pigmentary deposits are also common. The kidneys present more or less parenchymatous change with only moderate pigmentation. (*c*) In malarial cachexia the blood presents all the characteristics of an advanced anæmia, often distinguishable from pernicious anæmia only by the presence of the parasite. The spleen is greatly enlarged: it may weigh from seven to ten pounds. The organ is firm and resistant to the knife. The capsule is thickened and the parenchyma brownish or slate-colored, with areas of pigmentation. The liver presents less marked changes. It is of a grayish-brown or slate-color and shows extensive pigmentation. The kidneys are enlarged and of a grayish-red color. The peritoneum is thickened, opaque, and of a deep slate-color; the gastric and intestinal mucous membrane may have

the same hue. The gray matter of the brain is of a deep reddish-gray color or in very chronic cases a chocolate-brown. The meninges are congested. (*d*) Among accidental and late lesions is cirrhosis of the liver. Very extensive pigmentation may occur. Pneumonia is believed to be common; moderate albuminuria is frequent; acute nephritis is not uncommon; chronic nephritis may follow long-continued or repeated infection. In pernicious malaria the brain may show thrombosis, due to the parasites, with secondary softening of the surrounding tissue. The same thing may occur in the gastro-intestinal mucosa and be followed by superficial ulceration. For full information as to the important *rôle* played by the mosquito in the production of malaria, the reader is referred to the article by Professor Grassi in this number of the CLINICS.

MALTA FEVER.

DEFINITION.—A chronic, infectious, contagious disease, occurring most frequently in the Mediterranean region, and due to the *Micrococcus melitensis*. It consists of repeated febrile attacks followed in each case by a short afebrile period, and complicated by swelling of joints, anæmia, sweating, orchitis, and neuralgia.

ETIOLOGY.—Young and previously healthy adults who are unacclimated are most frequently attacked, and it is a serious disease in the British garrisons. The micrococcus is found in large numbers in the spleen.

MORBID ANATOMY.—The visceral changes are those common to all infectious diseases with high temperature. The small intestine is usually anæmic except in the upper part, where it may be intensely congested. The mesenteric glands show little change. The spleen is much enlarged, dark in color, and its pulp is soft and friable. The average weight is eighteen ounces. The liver is congested and its surface on section is pigmented. The kidneys are usually congested and may be slightly hemorrhagic. The agglutinative reaction can be obtained with the micrococcus and the blood of a patient affected with Malta fever. It should be remembered that this disease occurs in our new possessions, and that soldiers on their return home may bring the disease with them.

MEASLES.

ETIOLOGY.—A specific micro-organism, the identity of which is not yet definitely settled. Lesage describes a small micrococcus, obtained from the spleen in five cases, which grows best on simple agar.

MORBID ANATOMY.—The post-mortem appearances in measles are chiefly those of its complications and sequelæ. The skin, especially

about the face, may be swollen and slightly œdematous, and may show the remains of the characteristic rash, especially in the hemorrhagic type. Desquamation, when present, is in the form of fine branny scales. The gastro-intestinal mucosa is usually hyperæmic; Peyer's patches are frequently swollen, sometimes markedly so. The lungs invariably show evidence of bronchitis, and almost invariably lesions of bronchopneumonia with areas of collapse; less frequently lobar pneumonia may be found. The bronchial glands are invariably swollen. Pleurisy is less common. In debilitated infants severe stomatitis, cancrum oris, or ulcerative vulvitis may develop. In the middle ear catarrhal inflammation, which may go on to abscess formation, is not uncommon. Of the sequelæ tuberculosis is the most important; it is either miliary or a caseous pneumonia. Severe forms of conjunctivitis and ulcer of the cornea are not uncommon. Nephritis is exceedingly rare.

MUMPS.

Definition.—An acute, infectious, contagious disease, characterized by a marked cellular infiltration of the parotid glands, which do not tend to suppurate or to become fibroid.

Etiology.—(*a*) Probably a coccus infection. (*b*) Childhood and adolescence. Very young infants and adults are seldom attacked.

Morbid Anatomy.—Uncomplicated mumps is rarely fatal. Metastatic involvement of the reproductive organs and breasts is common. Of the complications meningitis, acute mania, endocarditis, gangrene, and optic atrophy are the most important.

MUSCULAR DYSTROPHIES.

Etiology.—(*a*) Traumatism. (*b*) Poisons,—*e.g.*, alcohol and lead. (*c*) Infectious diseases. (*d*) Lesions of nuclei in the motor path, which may be cortical, bulbar, or spinal.

Morbid Anatomy.—(*a*) Macroscopically there is first hypertrophy of the muscles with some increase in the connective tissue; later the muscles atrophy and show marked increase of fibrous tissue. (*b*) Microscopically the muscle is found to be first increased in size, with some increase of connective tissue; later the muscle-fibre is replaced by fat.

PANCREATITIS.

Etiology.—Practically unknown, though probably of a chemical nature. Acute pancreatitis can be produced in the lower animals experimentally by the injection of an artificial gastric juice, but it is impossible to tell which form of the disease will result. *Classification.*

—I. Acute. (*a*) Hemorrhagic. (*b*) Suppurative. (*c*) Gangrenous. II. Chronic.

Morbid Anatomy.—(*a*) In hemorrhagic pancreatitis the organ is enlarged, thin, irregular in outline, intensely congested, coagulated blood often appearing on the surface of the pancreas while other areas may be comparatively normal. The organ is somewhat harder than normal, but may be almost diffluent. There may be adhesions to the surrounding structures. The omentum and mesentery may sometimes show focal areas of fat necrosis, whereas the serous membranes and the subcutaneous tissue are often the seat of various-sized hemorrhagic extravasations. The stomach may contain grumous blood and show the evidences of an acute gastritis. (*b*) Suppurative pancreatitis may follow the hemorrhagic variety or may be the result of septic infection from a distant focus. The organ appears swollen, is softer in consistence, and may be the seat of a single abscess or of numerous ones. It is more or less adherent to the surrounding structures, and it is exceedingly difficult to separate it from its adhesions. There may be numerous metastatic abscesses in other organs. (*c*) In gangrenous pancreatitis the organ may present a dry necrotic appearance, but, as a rule, it is a dark slate-colored, diffluent mass, surrounded by more or less dense adhesions with the surrounding structures. Sometimes it has been found that the organ has entirely disappeared, its place being taken by an abscess-cavity containing a foul-smelling mass.

PARASITES.

Pediculi.—(*a*) *Pediculus Capitis.*—The female measures from one and eight-tenths millimetres to two millimetres in length, the male being somewhat smaller. The darker the skin of the person infested the darker is the color of the parasites. So marked is the peculiarity that some writers are of the opinion that different species affect different races. The ova are white glistening specks firmly adherent to the hair. Considerable irritation is caused by these animals, and when this is severe the hair on the back of the head may be found matted with soft yellow crusts. The scalp is covered with moist red granulations. The cervical lymphatic glands posteriorly are enlarged. This condition is most frequently seen in children. (*b*) *Pediculus Pubis.*—It differs slightly from the above in that it is smaller and infests regions, as the axillary, the pubic, and the periocular, where the hair is short. (*c*) *Pediculus corporis* is the largest form of the parasite. It lives entirely in the clothing. By its constant irritation it causes dermatitis, and, if present for a long time, pigmentation and thickening of the skin.

Cimex Lectularius (common bedbug). (*a*) *Pulex Irritans* (the common flea). (*b*) *Pulex Penetrans* (sand-flea, jigger). The latter is common in tropical and subtropical countries. It is smaller than the common flea. It burrows under the skin and produces a pustular swelling.

Sarcoptes (*Acarus*) *Scabiei*.—The female itch-mite is .45 of a millimetre long and .35 of a millimetre broad; the male is about one-half the size. Its color is pearly white. The burrow in the skin is about one centimetre in length, and is present where the skin is moist, as in the webs of the fingers and toes. Cutaneous lesions result from the scratching instigated by the irritation caused by the parasite.

Cestodes.—*Intestinal Cestodes*.—(*a*) *Tænia solium* in the mature form may reach to twelve feet or even more in length. It is composed of numerous segments about one-third of an inch long and averaging a fourth of an inch wide. The head is very minute, being no larger than the head of a pin. In front is a rostellum and at the base of this is a fringe of hooklets. It has four suckers. The worm is hermaphroditic. When mature thousands of ova are passed by the rectum. The embryo has six hooklets. It penetrates the walls of the stomach and burrows into the tissues of the animal that has swallowed it. (*b*) *Tænia Saginata* is larger, longer, and of more frequent occurrence than the preceding. The head is nearly square and measures more than two millimetres in breadth, but has no hooklets. The segments are larger than those of the *Tænia solium*. The reproductive organs are on the ventral aspects of the segments in the median lines. (*c*) The *Bothriocephalus latus* is larger and longer than any of the flat worms. In the mature state it is twenty-five feet or more in length. It has no hooklets, but is furnished with slit-like fossæ on the head, which act like suckers. The larvæ develop in the peritoneum of fish. (*d*) *Tænia flavo punctata* is very rare. It is about sixteen centimetres long. (*e*) The *Cysticercus cellulosæ* is the larval form of the tænia solium. It is found in the muscles, brain, cord, peritoneum, or almost any other tissue of the affected animal. The surrounding capsule is frequently calcified. In the making of many autopsies it is surprising how few tænia are found in the intestinal tract. My experience is limited to but two cases. One of these was that of a man who committed suicide with opium. Two *Tænia saginata* were found, the head of the first one being firmly attached beneath a fold of one of the valvulæ conniventes high up in the jejunum and the other five or six feet further down the intestine, the segments of both worms then continuing on down to near the ileo-cæcal valve.

Nematodes.—(*a*) *Ascaris Lumbricoides*.—It is a cylindrical worm

with both ends pointed. The female is from ten to sixteen inches in length, the male considerably smaller. It is brownish yellow, reddish, or white in color. The head ends in three lips. (*b*) The *Oxyurus vermicularis* (seat-worm) is a very small round worm, about ten millimetres long. (*c*) The *Trichina Spiralis* in the mature state lives in the intestine; in the immature state in the muscles. The embryo is surrounded by a capsule, which quickly calcifies. Under the microscope the embryo can be seen coiled up in its capsule; it is less than a millimetre in length. (*d*) The *Anchylostomum duodenale* lives in the upper part of the intestine. The female is the larger, and varies from ten to sixteen millimetres in length. At the anterior portion of its head are hooklets, with which it attaches itself to the intestinal walls. It is frequently associated with Egyptian chlorosis. (*e*) The embryo of the *Filaria sanguinis hominis* is a round worm one-seventy-fifth to one-one-hundredth of an inch long. It is enclosed in a delicate sac. It circulates freely in the blood, but only at night. The adult parasite is located in the lymphatic vessels and is three or four inches in length. According to Manson, it is introduced into the body by the mosquito.

Distomiasis.—*Classification.*—(*a*) Liver-flukes. (*b*) Blood-flukes. These worms are lanceolate in shape, quite flat, and possess a distinct head and neck. They are three-fourths of an inch long and about half an inch broad. The color is dull brown. The female blood-fluke has a grooved channel posteriorly for the reception of the male. They have two suckers, one near the mouth and the other near the ventral portion of the body. The liver-fluke infests the upper intestine and the bile-ducts. It causes the "liver-rot" in sheep. The blood-fluke is found chiefly in the portal system and the veins of the bladder. The ova may be seen in the urine as elongated ovoid bodies, sharply pointed at one extremity, and containing black pigment. They can easily be seen with a low power of the microscope.

Myiasis.—By this term is meant a condition in which a diseased part becomes "living," as it is called. It is caused by the larvæ of certain flesh-flies, of common house-flies, or of the bot-flies of oxen or sheep. The ova of these flies may be deposited in the nostrils, ears, conjunctiva, open wounds, or even in the vagina during the puerperium.

Echinococcus Disease.

Definition.—A parasitic disease, found most frequently in those countries, as Iceland and Australia, where the dog lives in intimate association with man; it is characterized by the formation of endogenous or exogenous cysts in various portions of the body.

Etiology.—The *Tænia echinococcus,* a very small, thread-like tape-

worm (length from three to six millimetres), having only three segments. The head has four suckers, a rostellum, and a double row of hooklets. The adult worm is found in the dog. The embryos (scolices) are found in the ox, hog, sheep, horse, and man. *Classification.*—According to the manner in which the cysts grow, they are called (*a*) endogenous, (*b*) exogenous, (*c*) multilocular. *Distribution in Man.*—(*a*) Liver (most common). (*b*) Lung and pleura. (*c*) Intestinal tract. (*d*) Kidney, etc.

Morbid Anatomy.—The embryo, freed from the cyst by digestion in the stomach, burrows through the intestinal wall and is carried to various organs: it then loses its hooklets and is gradually converted into a cyst (hydatid) having two walls, external laminated, internal granular or parenchymatous, containing blood-vessels and muscle-fibres. The interior is filled with a clear non-albuminous fluid, specific gravity 1005-1009, usually containing sugar and hooklets. From irritation of surrounding tissues a fibrous capsule generally develops on the outside. The cysts vary in size from that of a small pea to that of a child's head. From the inner (parenchymatous) layer may develop brood capsules, which in their turn produce numerous scolices. The cyst grows slowly; when the embryo dies, the whole becomes calcified. Sometimes the cysts suppurate; occasionally they rupture into adjacent structures.

PERITONEUM, CANCER OF.

Etiology.—(*a*) Female sex. (*b*) Age from forty to sixty years. (*c*) Heredity. (*d*) Secondary to cancer of stomach or ovaries most often.

Morbid Anatomy.—Almost always secondary. Often spoken of as "miliary carcinosis," because nodules are small, spherical, and diffuse. The serous membranes are pale, thickened, with marked fibrinous deposits, which form adhesions to neighboring viscera: the omentum is indurated and forms a mass transversely across the abdomen; the bowels are often firmly matted together. Ascites is usually present; the amount of fluid may be only a few ounces or several pints. In some cases of colloid cancer the masses are of large size.

PERITONITIS, ACUTE GENERAL.

Etiology.—(*a*) Exposure to cold and wet. (*b*) The following micro-organisms have been found: *Streptococcus pyogenes*, *Bacillus coli communis*, *Staphylococcus aureus*, *Micrococcus lanceolatus*, *Bacillus proteus*, and *B. pyocyaneus*; rarely are found the gonococcus and the bacilli of anthrax and typhoid. (*c*) Perforation of the bowel. *Classification.*—(*a*) Serous. (*b*) Serofibrinous. (*c*) Fibrinous. (*d*) Purulent. (*e*) Putrid. (*f*) Hemorrhagic.

Morbid Anatomy.—In acute general peritonitis the peritoneum has lost its lustre, is opaque, and is covered with an exudate varying with the type of the disease. The intestinal coils are distended and glued together with lymph. They are more or less displaced and compressed and their walls are easily torn. The serous membrane may be easily separated from the muscular coat. In peritonitis due to perforation the peritoneum and its contents are discolored by the fæces, while the peritoneal cavity contains gas, which escapes with a hissing noise when it is opened.

PERITONITIS, CHRONIC.

Etiology.—(*a*) Follows acute. (*b*) Tuberculosis. (*c*) Extension of inflammation from contained organs. (*d*) Cancer. *Classification.*—(*a*) Local adhesion. (*b*) Diffuse adhesive. (*c*) Proliferative. (*d*) Hemorrhagic.

Morbid Anatomy.—(*a*) Localized peritonitis occurs about the spleen, liver, intestines, mesentery, and pelvic organs. Bands of connective tissue more or less firmly organized bind the various organs together, producing marked alterations in appearance and position of the parts. The peritoneum is thickened and puckered. (*b*) Diffuse adhesive peritonitis follows acute inflammation, either simple or tubercular. The abdominal cavity is often obliterated; the intestinal coils are firmly matted together by the plastic exudate, which becomes eventually converted into bands of fibrous tissue. The liver and spleen are usually involved in the adhesions. (*c*) In the proliferative form there is great thickening of the peritoneum, which is opaque and white in color. The omentum is usually rolled into a thick mass between the stomach and the colon. The liver and spleen are the subjects of a chronic capsular inflammation; both are usually smaller in size, with thickened, wrinkled capsules. There are seldom many adhesions, and serous effusion may be present in the abdominal cavity. The intestinal wall is greatly thickened and the mucous membrane of the ileum is thrown into folds. Nodular thickenings may be present and be mistaken for tubercles. (*d*) The hemorrhagic form occurs particularly in cancerous and tubercular conditions. Layers of new connective tissue form on the surface of the peritoneum; they contain large blood-vessels, from which the bleeding occurs. It is commonly a circumscribed process.

PLAGUE.

Definition.—An acute infectious, contagious epidemic disease due to the *Bacillus pestis*, occurring usually in the far East, and characterized by marked glandular enlargements which tend to suppuration and by a general septic condition.

Etiology.—A bacillus discovered by Kitasato. It is a short rod with rounded ends, and is found in the blood, glands, and viscera. Hossack found no buboes in thirty per cent. of his cases in Calcutta in 1900. *Varieties.*—(*a*) Bubonic. (*b*) Pneumonic. (*c*) Septic. (*d*) Intestinal. (*e*) Meningeal. (*f*) Carbuncular.

Morbid Anatomy.—(*a*) At the point of inoculation, which usually occurs on the lower extremities, there appears a small spot (plague-corpuscle) which soon becomes a vesicle and then a pustule. (*b*) Following primary inoculation, the inguinal glands become swollen succeeded in order by the axillary, cervical, popliteal, and then any of the glands in the body may become affected. The diseased glands swell rapidly and are at first tense and firm to the touch, but soon undergo a suppurative change, and in rare cases gangrene ensues. (*c*) Carbuncles may develop in the skin of the legs, hips, and back. Subcutaneous hemorrhages are very common and may also occur from the mucous membranes. (*d*) The central nervous system, especially the brain, is deeply congested. The brain substance may become softened and the blood-vessels, especially the veins, are engorged. (*e*) The lungs are deeply congested, especially posteriorly. (*f*) The pericardium contains an excess of blood-stained fluid. The right heart is dilated with black, imperfectly coagulated blood, and the whole venous system is engorged. The heart-muscle is pale and somewhat softened. (*g*) The stomach and small intestine contain blood or blood-stained fluid. There may be ulceration, but Peyer's patches are not affected. The spleen is greatly enlarged in all cases. (*h*) The dorsum of the tongue is coated, but the edges, the tip, and often the median raphe remain pink and clean; sometimes, however, becoming red and dry (Hossack). The disease must be distinguished from puerperal fever, septicæmia, pyæmia, smallpox, influenza, cerebro-spinal meningitis, diphtheria, erysipelas, measles, gonorrhœa, syphilis, mumps, malaria, scrofulous glands, Hodgkin's disease, etc. In a recent case of a Chinaman suspected of having the plague, the writer found almost complete occlusion of the prepuce, with a discharge containing the gonococcus, and in the suppurating bubo a fat diplo-bacillus which did not stain by Gram's method.

POISONING.

Definition.—A poison is any substance which, when taken into the system and either being absorbed or by its direct chemical action upon the parts with which in contact, or when applied externally and entering the circulation, is capable of producing deleterious results. (Wormley.)

Etiology.—Poisoning commonly results from alcohol; morphine; lead; arsenic; food (bromatotoxismus); meat poisoning (kreotoxismus); milk products (galactotoxismus); fish and shell-fish (ichthyotoxismus, mytilotoxismus); grain poisoning (sitotoxismus), three kinds,—ergotism, lathyrism, and pellagra.

Alcoholism, Chronic.

Morbid Anatomy.—There are no really characteristic lesions. I. *Gastro-intestinal Tract.*—(*a*) Chronic hypertrophic gastritis may be followed by (*b*) atrophic gastritis with dilatation. (*c*) Hypertrophic or atrophic scirrhosis of the liver. II. *Vascular System.*—(*a*) The heart is usually enlarged and its muscle often thin, fatty, and friable. (*b*) The blood-vessels are frequently sclerosed, especially those arteries exposed to much strain. (*c*) The venules of the cheek and nose are often distended. III. *Central and Peripheral Nervous System.*—(*a*) The piarachnoid is thickened, with wasting of its convolutions. (*b*) The blood-vessels are thickened, tortuous, and may show miliary aneurisms. (*c*) The motor nerves of the muscles are sometimes altered (multiple neuritis). IV. *Genito-urinary Tract.*—(*a*) The kidneys are enlarged, cyanotic, and indurated. (*b*) The bladder is thickened and often shows signs of chronic cystitis.

Arsenical Poisoning.

Classification.—(*a*) Acute. (*b*) Subacute. (*c*) Chronic.

Morbid Anatomy.—I. In acute arsenical poisoning there is a marked gastro-enteritis, which differs in severity according to the amount taken. The mucous membranes are intensely swollen, œdematous, and present small emphysematous bullæ or diphtheritic exudate. Pustular eruptions may occur in both the stomach and intestines. The contents of the stomach are usually of a brownish color. II. (*a*) In subacute arsenical poisoning, or where large doses have been taken, patches varying in size from a dime to a silver dollar, consisting of an opaque white, yellowish, or even violet coagulated lymph mixed with arsenous acid and firmly fixed to the mucous membrane, with signs of intense inflammation around them, may be found in the intestines. (*b*) White spots of arsenic are sometimes discovered between the rugæ, and fatty degeneration of the intestinal epithelium and of the viscera is also present. III. (*a*) Chronic arsenical poisoning is characterized by wide-spread fatty degeneration affecting especially the heart, liver, spleen, and kidneys. (*b*) Marked changes are also found in the voluntary muscles, which show wasting, fatty degeneration, and often cirrhosis. (*c*) Trophic changes are common, such as overgrowth of hair

and nails, both of which are harsh and brittle. The skin is harsh, dry, and frequently shows eruptions. (*d*) Sufficient arsenic for purposes of identification usually remains in the body even though the elimination is rapid.

Carbolic Acid Poisoning.

Morbid Anatomy.—The mucous membranes of the alimentary tract or bronchial tubes show intense injection. In the mouth, œsophagus, and stomach are numerous white spots with occasionally a dark centre and inflammatory border due to the local action of the poison. The liver, kidneys, and spleen may be congested and filled with a dark fluid. The urine may be dark in color.

Carbon Monoxide Poisoning.

Morbid Anatomy.—After death the blood retains its bright cherry color for some time, and when shaken forms a froth of a violet color. The skin and internal organs, as also the patches of post-mortem congestion, exhibit a bright red color. The lungs are frequently congested. Another poison may be taken with suicidal intent, the gas then turned on, and the patient die from the first poison.

Cocaine Poisoning.

Morbid Anatomy.—At post mortem the heart is found in diastole and the nerve-centres are said to be congested. Cocaine should be tested for before making the diagnosis.

Ergot Poisoning.

Morbid Anatomy.—After death from ergot poisoning the arteries are found contracted and the abdominal viscera inflamed. In the chronic form the posterior columns of the cord are sclerosed and microscopical sections resemble those characteristic of locomotor ataxia.

Lead Poisoning.

Classification.—(*a*) Acute. (*b*) Chronic.

Morbid Anatomy.—I. (*a*) In acute lead poisoning there is marked gastro-enteritis, and the bowels usually contain a large amount of blackish fluid. (*b*) The kidneys show evidence of acute diffuse nephritis. II. (*a*) In chronic lead poisoning the distinctive features are a marked fatty degeneration affecting the muscles, kidneys, spleen, and liver. There is often marked cirrhosis with atrophy of these organs. (*b*) Arteriosclerosis with hypertrophy of the heart is also marked. (*c*) Distinct gouty deposits are often found, particularly about the big toe. (*d*) The brain is sometimes shrunken and dry, the blood-vessels being

constricted; or these organs may be pale and extremely firm, or pale and œdematous, as in cases of uræmia. (*e*) The small intestines may show areas of extreme contraction.

Mercurial Poisoning.

Morbid Anatomy.—(*a*) The mucous membranes of the gastro-intestinal tract, especially the small intestine and cæcum, show extensive desquamation with hyperæmia and ecchymoses. The bowel generally contains large quantities of liquid of a yellowish-brown or blood-stained character. (*b*) In some acute cases decalcification of the bones occurs, with a deposit of lime.

Opium Poisoning.

Classification.—(*a*) Acute. (*b*) Chronic.

Morbid Anatomy.—I. (*a*) In acute poisoning there is nothing to distinguish the condition of the brain from that of other cases of cerebro-congestion. (*b*) Extreme passive congestion of the bases of the lungs may take place as in cerebro-apoplexy (Osler). II. Cases of uncomplicated chronic poisoning are rare. The most important lesion is fatty degeneration of the heart. The liver may show similar changes.

Pellagra Poisoning.

The lesions found are in the posterior columns and the crossed pyramidal tract. The cells in the anterior horn are deeply pigmented, and pigment is found in the internal organs and the skin. The brain presents general wasting; the ventricles are somewhat distended and contain an excess of fluid.

Phosphorus Poisoning.

Classification.—(*a*) Acute. (*b*) Chronic.

Morbid Anatomy.—I. (*a*) In acute phosphorus poisoning the gastro-intestinal tract, especially in the stomach, shows an intense degree of inflammation. Hemorrhages are common and the stomach may contain grumous (coffee-ground) blood. The mucous membrane is the seat of numerous ecchymoses as well as more or less extensive necroses. (*b*) The skin, the serous membranes, the muscles, and the adipose tissues all show numerous small hemorrhages. (*c*) The blood is liquid and dark. (*d*) The skin is jaundiced. (*e*) The liver, in the early stages increased in size, soon becomes small (from one-half to one-third of the normal bulk), the capsule is wrinkled and shrunken, the color is pale yellowish, and on section the organ presents yellowish patches in the midst of which are areas of deep

congestion. Drops of fat are seen upon the knife. (*f*) The kidneys are large, their cortex pale, and the medullary portions congested. The epithelium often shows marked granular degeneration. (*g*) As a rule, the spleen is not markedly altered. II. (*a*) In chronic poisoning by phosphorus wide-spread fatty degeneration is the rule. (*b*) In cases of workers in phosphorus having defective teeth, necrosis of the jaw is not uncommon.

Snake Poisoning.

Classification.—(*a*) Cobra. (*b*) Australian snakes. (*c*) Viperine snakes.

Morbid Anatomy.—I. (*a*) After death caused by cobra bite rigor mortis occurs as usual. (*b*) The areolar tissue in the region of the bite is infiltrated with a pinkish fluid and the vessels are injected. (*c*) The blood presents no demonstrable change. (*d*) The veins of the pia mater are usually engorged, and the ventricles often contain turbid fluid. (*e*) The lungs are generally congested and the lining of the bronchi injected. (*f*) The appearance of the kidneys varies from normal to one of intense congestion. II. (*a*) After death following the bite of an Australian snake the appearances are much the same as those just described. (*b*) The blood may contain soft coagula, (*c*) the lungs are sometimes the seat of hemorrhages, and the mucous membranes may be intensely congested and hemorrhagic. (*d*) The central nervous system shows engorgement of the blood-vessels. III. (*a*) At autopsy, after the bite of a viperine snake, the region of the wound is seen to be the seat of intense œdema and extravasation of blood, and the underlying muscles are frequently disorganized and even diffluent from the latter cause. (*b*) Hemorrhages may also be found in any of the organs and along the alimentary tract. (*c*) The kidneys are acutely congested or hemorrhagic. (*d*) The blood is fluid.

PSOROSPERMIASIS.

Definition.—A condition produced by the presence of oval, transparent bodies belonging to the coccidia, to which class the malarial organism also belongs. *Classification.*—(*a*) Internal. (*b*) Cutaneous.

Morbid Anatomy.—I. (*a*) In the majority of cases of the internal form the psorosperms have been found in the liver. (*b*) Whitish growths resembling tubercles and containing the coccidia have been found upon the peritoneum, omentum, and pericardium. (*c*) Similar masses are sometimes seen in the ileum, liver, spleen, and kidneys. The liver may be enlarged and contain caseous foci which are surrounded by areas of congestion. (*d*) The spleen may be similarly affected. II.

(*a*) In cutaneous affections the lesions closely resemble those of tuberculosis of the skin. (*b*) A case has been reported in which at autopsy nodules were found in the lungs, adrenals, testicle, spleen, on the surface of the liver, and on the pleuræ. Great numbers of psorozoa were found in the lesions. (*c*) Successful inoculations were made into rabbits and dogs.

PULMONARY STENOSIS; PULMONARY INSUFFICIENCY—see HEART.

PURPURA.

DEFINITION.—A disease characterized by the appearance on the skin of numerous blotches of extravasated blood and by great debility.

ETIOLOGY.—Very little is known of its origin. It is apparently more common in males than in females and more frequent in the young than in the old. *Classification.*—The forms are (*a*) Purpura simplex, (*b*) purpura hemorrhagica, (*c*) purpura rheumatica, (*d*) iodic purpura, (*e*) Henoch's purpura.

MORBID ANATOMY.—(*a*) Autopsy reveals simply the existence and extent of hemorrhagic effusions with evidences of anæmia. (*b*) Occasionally the skin presents erosions or ulcerations. (*c*) The hollow viscera and serous cavities may contain considerable quantities of blood-stained serum. (*d*) The serous membranes and solid organs, as well as the skin, may reveal hemorrhages varying in size from a pin's head to the palm of the hand. (*e*) Congestion and œdema of the lungs are frequently present. (*f*) Slight degrees of acute diffuse nephritis may occur. (*g*) Ulcerations of the intestines with enlargement of the solitary and agminated glands are sometimes present.

RAYNAUD'S DISEASE.

NOTE.—See Dr. Graham's article in this number of the CLINICS for a description of this condition.

RELAPSING FEVER.

DEFINITION.—An acute, epidemic, contagious disease, not found at the present time in America unless imported, occurring in the same class of persons as typhus fever, giving rise to a fever which lasts from five to seven days, followed by relapses, and due to the *Spirochætæ* of Obermeier, which are found in the blood only during the paroxysms of fever.

ETIOLOGY.—A specific, motile organism.

Morbid Anatomy.—No characteristic or constant lesions are found after death. The following are sometimes present. (*a*) If death occurs during the paroxysm, the spleen is large and soft; the pulp is purple. The follicles are enlarged and often obliterated, though they may be gray or whitish yellow in color. Infarcts are not uncommon. (*b*) The heart is flabby, of a pale dirty-gray color, and very friable. (*c*) The liver is more enlarged in this than in any other infectious fever. Its color is a uniform gray-red. Fatty degeneration may be marked. (*d*) The kidneys may retain their normal weight. The renal parenchyma is soft and flabby; the cortical substance is increased and shows cloudy swelling. Hemorrhagic spots or lines radiating to the pyramids are often observed. (*e*) The lungs may be the seat of pneumonic infiltration, bronchitis, or bronchiectasis. (*f*) Hyperplasia of the bone marrow has been found. *Complications.*—(*a*) Pneumonia is frequent. (*b*) Rupture of the spleen. (*c*) Nephritis and hæmaturia. (*d*) Ophthalmia in certain epidemics. (*e*) Abortion.

RETINITIS.

Etiology.—(*a*) Bright's disease. (*b*) Syphilis. (*c*) Leukæmia. (*d*) Advanced anæmia. (*e*) Diabetes. (*f*) Disease of vascular system. *Classification.*—(*a*) Albuminuric. (*b*) Diabetic. (*c*) Syphilitic. (*d*) Leukæmic. (*e*) Septic. (*f*) Hemorrhagic (vascular).

Morbid Anatomy.—(*a*) Examination of the retina reveals hemorrhages in the layer of nerve-fibres and the development of opacities. (*b*) The hemorrhages vary greatly in amount; when recent their color is bright red, but gradually becomes almost black. (*c*) The opacities are due to a fibrinous exudate in or to fatty degeneration of the retinal elements. (*d*) In the albuminuric form white spots forming stellate figures occur in the macular region. (*e*) There may be cloudiness of the retina, caused by the effusion of serum.

RHEUMATIC FEVER.

Etiology.—(*a*) Exposure to cold and wet. (*b*) Usually regarded as a coccus infection, though a bacillus has also been described as the etiologic factor.

Morbid Anatomy.—(*a*) The affected joints are swollen, tense to the touch, and somewhat hyperæmic. The fluid in the joint is turbid, and contains albumin, leucocytes, and a few flakes of fibrin, but rarely pus. There may be slight erosion of the cartilages. (*b*) Endocarditis occurs in about sixty per cent. of all cases. The verrucose variety is most common. The mitral valve is most frequently involved. (*c*) Pericarditis may occur, with or without endocarditis. It may be fibrin-

ous, serofibrinous, or, in children, purulent. (*d*) Myocarditis occurs most frequently in association with endopericarditis. It leads to weakening and dilatation of the heart-muscle, and is the most common cause of sudden death in rheumatic fever. (*e*) Pleurisy and pneumonia occur in about ten per cent. of all cases. (*f*) Rheumatic nodules, varying in size from a small shot to a large pea, are found on the fingers, hands, and wrists. They may also occur about the elbows, knees, spines of the vertebræ, and scapulæ. (*g*) Meningitis is extremely rare. (*h*) Purpura may be present.

RHEUMATISM, CHRONIC.

Morbid Anatomy.—(*a*) The synovial membranes are injected. There is usually not much effusion. The capsules, ligaments, and sheaths of the tendons are thickened. There may be erosion of the cartilages. As a result of these changes, the joints are often deformed and ankylosis may occur. (*b*) Muscular atrophy, especially about the joints, frequently follows. (*c*) Valvular heart-lesions, due to sclerotic changes, are of common occurrrence.

RICKETS.

Morbid Anatomy.—(For bone changes, see page 208.) (*a*) The muscular structures show much atrophy, both microscopically and macroscopically. (*b*) The mucous membranes are subject to catarrhal processes with marked hyperæmia. (*c*) The lungs and heart often present changes due to deformity of the bony structures of the chest. The histological preparation of a slide of one of the nodes on the ribs forms one of the prettiest microscopical pictures imaginable. (See illustration in Ziegler's "Pathology.")

RUBELLA (RÖTHELN, GERMAN MEASLES).

Morbid Anatomy.—The disease is never fatal in uncomplicated cases. There are no distinctive lesions other than the rash, which may fade entirely after death.

SALPINGITIS.

Etiology.—(*a*) Infection by micro-organisms,—*e.g.*, the *Gonococcus,* the *Streptococcus,* the *Bacillus coli communis,* etc. *Classification.*—(*a*) Hydrosalpinx. (*b*) Pyosalpinx. (*c*) Hæmatosalpinx. (*d*) Proliferous salpingitis.

Morbid Anatomy.—(*a*) In acute salpingitis the Fallopian tubes are swollen. The neighboring blood-vessels are dilated, tortuous, and overfilled with blood. There is often considerable exudate on the serous

surfaces, causing adhesions of the tubes to the surrounding structures. On section the lumen of the tubule is found to contain serous, muco-purulent, or hemorrhagic fluid. The mucous membrane is thickened, swollen, and often intensely congested. To show the ciliated cells, though these may have been shed by the inflammatory process, care must be taken to harden the tissue at once after removal and in the same manner as to show karyokinesis. (*b*) In chronic (proliferous) salpingitis the tubes become enormously thickened, hard, and resistant to the touch. The adhesions to surrounding tissues are very marked and more or less completely organized. The new connective tissue contracts, throwing the organs out of their proper relation and often obliterating the normal appearance of the organs.

SAPRÆMIA.

Definition.—By sapræmia is understood the symptom-complex produced by the presence in the blood and tissues of the vital chemical products known as toxins. These toxins may be formed by the action of pyogenic or saprophytic micro-organisms.

Morbid Anatomy.—(*a*) Rigor mortis comes on early and soon passes away. Decomposition sets in rapidly and often interferes with the examination of the body. Hypostatic congestion is very marked, the blood flowing readily on section with the knife. (*b*) Eight or ten hours after death the organs are diffluent or distended with fetid gases. (*c*) Serous membranes throughout the body, especially those of the pleuræ, pericardium, and blood-vessels, show extravasations of dark blood. (*d*) The liver, spleen, and kidneys are swollen, congested, and hemorrhagic. The splenic pulp is sometimes almost diffluent.

SCARLET FEVER.

Etiology.—(*a*) The majority of cases occur before the tenth year. (*b*) Infants and adults are usually exempt. (*c*) Cocci are frequently found in the throat-lesions and in the blood. Class, of Chicago, claims to have isolated a specific coccus, which has also been described by Baginsky.

Morbid Anatomy.—(*a*) Rigor mortis is usually well marked. Decomposition may set in early and develops with exceptional rapidity, cadaveric lividity usually appearing before death. (*b*) The blood is dark in color, thin, and coagulates imperfectly. The vessel-walls are usually stained. (*c*) Except in the hemorrhagic form the skin after death rarely shows a trace of the rash. (*d*) In the throat follicular tonsillitis, diphtheritic membrane, or suppuration may be present. Punctate hemorrhages, especially about the mouth, are always observed.

(*e*) Catarrhal inflammation of the gastro-intestinal mucous membrane is not uncommon. (*f*) In severe cases an intense lymphadenitis, with much inflammatory œdema, is found in the neck. This may lead to suppuration or even gangrene, and in rare cases to ulceration of the carotid artery and fatal hemorrhage. (*g*) The kidney lesions are most important. Acute diffuse nephritis is present in a majority of cases. It is frequently of the glomerular type. This lesion is not infrequently followed by chronic parenchymatous nephritis. (See Kidney, page 247.) (*h*) Endocarditis, which may be either simple or malignant, is not infrequent. Pericarditis and myocardial changes are less common. (*i*) The spleen is often enlarged, and shows the changes which characterize acute splenic tumor. (*j*) Hemorrhages into the subserous tissues beneath the pericardium, endocardium, and pleura are quite frequent. *Complications.*—(*a*) The most important is nephritis. The urine is small in quantity, of a high specific gravity, cloudy, and of a dark blood-color. It contains large amounts of albumin, free blood, and epithelial cells, with hyaline and epithelial tube-casts. Œdema may be slight or marked; in a few cases œdema of the glottis has caused sudden death. (*b*) Heart complications are next in importance. There may be endocarditis, pericarditis, or myocarditis. (*c*) Catarrhal pneumonia, more rarely croupous pneumonia or pleurisy, may occur. (*d*) Involvement of the ear may lead to thrombosis of the lateral sinus, meningitis, abscess of the brain, or necrosis *en masse* of the middle ear. (*e*) Adenitis may result. The glands of the neck are those most frequently involved. There may be great destruction and loss of tissue. (*f*) Arthritis of a rheumatic type or more closely resembling the gonorrhœal variety may be found. In the latter affection only one joint is involved and suppuration may supervene. I have made a number of post mortems on these cases, and have found no pathognomonic lesions. The toxin seems to act especially on the epithelial cells. In one of my cases death occurred in convulsions twenty-four hours after the onset of vomiting and without the appearance of any rash. The diagnosis was confirmed by a sister being attacked with the disease later on.

SCLERODERMA (HIDE-BOUND SKIN).

Morbid Anatomy.—(*a*) *Circumscribed Form.*—On the skin are found patches varying in size and of a waxy or dead white appearance. They are brawny, hard, and inelastic. (*b*) *Diffuse Form.*—This form usually occurs in the extremities or on the face. Gradually a diffuse brawny induration develops. The skin becomes firm, hard, and so closely united to the subcutaneous tissue that it cannot be picked up or

pinched. The color may be natural. The skin is commonly glossy, drier than normal, and unusually smooth.

SCURVY.

Etiology.—(*a*) Deficiency of fresh vegetables. (*b*) Bad hygienic surroundings.

Morbid Anatomy.—(*a*) After death decomposition sets in rapidly. The hemorrhagic patches observed in the skin during life are soon obscured by post-mortem lividity. (*b*) The subcutaneous tissues, especially those of the lower extremities, contain a blood-stained fluid with here and there discolored patches, some black and others of a pale color. In one of my cases these patches were mistaken for bruises, and a husband was arrested for causing the death of his wife by violence. (*c*) About the back of the thigh and knee the muscles and tendons may be embedded in a thick, firm clot, and themselves contain numerous hemorrhagic foci. (*d*) The blood is dark and fluid. Hemorrhages may be present in any of the serous membranes or internal organs. (*e*) The gums are swollen, sometimes ulcerated, and the teeth may have fallen out. (*f*) Hemorrhages in the mucous membranes are extremely common. Rarely there may be ulcers in the intestines. (*g*) The spleen is enlarged and soft, while fatty changes are constant in the liver, kidneys, and heart. (*h*) There is very little wasting of the subcutaneous fat or of the muscles.

SCURVY, INFANTILE.

Etiology.—Usually associated with improper food, such as malted or condensed milk. Cases, however, have been reported in breast-fed children.

Morbid Anatomy.—(*a*) The most important lesions are increased vascularity and extravasation of blood affecting the periosteum and bones, especially those of the lower limbs. Extensive hemorrhages are frequently found between the periosteum and the bone. They may also occur in the cavity of long bones, forming masses of blood-clots. (*b*) These deep-seated extravasations may give rise to muscular swellings and in some cases to extravasations in the joints. (*c*) Smaller extravasations have been observed in the pleura, lungs, spleen, intestines, and kidneys. (*d*) Fractures are not uncommon. In fact, in the majority of cases there are bone changes analogous to those of rickets. (*e*) The gums are spongy, sodden, distended with serum, and sometimes covered with blood. (*f*) One of the most characteristic lesions is extravasation of blood into the orbital cavity, causing displacement of the eyeball downward and forward. The report of the Ameri-

can Pediatric Society on this condition is of a most interesting character.

SEPTICÆMIA.

Definition.—A condition produced by the presence of pyogenic micro-organisms in the blood and tissues without areas of suppuration.

Morbid Anatomy.—(*a*) There are no characteristic lesions. The spleen is enlarged, dark in color, and soft in consistency. (*b*) Hemorrhages are common, especially on the serous surfaces. (*c*) No thrombi, emboli, or abscesses are found. (*d*) Various organs, such as the liver, may show microscopic or macroscopic areas of focal necrosis. By this term is meant a minute area of necrotic change produced in the viscera or in the muscles by the presence of bacteria. (This condition may possibly be produced by toxic agents independently of bacteria.) Microscopically there are three zones,—first, an inner or bacterial zone mainly composed of bacteria and leucocytes; secondly, a pale, poorly sustained area in which the cells have undergone marked granular change; thirdly, an outer zone of inflammatory reaction containing leucocytes, proliferated cells, and possibly a small amount of fibrin.

SEPTICÆMIA, PUERPERAL.

Morbid Anatomy.—(*a*) In some cases no lesion can be discovered except an abnormally thin condition of the blood with beginning inflammation in the veins and lymphatics of the uterus. (*b*) As a rule, the vagina is inflamed and œdematous. (*c*) Perineal lacerations, if present, are swollen and coated with a dirty yellowish membrane. (*d*) The endometrium is covered with purulent *débris* or may be necrotic. (*e*) The peritoneal cavity is filled with an offensive serous fluid. (*f*) The intestine is covered with a fibrinous exudate and may be enormously distended with gas.

SEPTICOPYÆMIA.

Definition.—Septicopyæmia is the condition produced by pyogenic micro-organisms and metastatic or pyæmic abscesses in the tissues and organs of the body.

Etiology.—The following organisms are usually associated with the above condition: streptococcus, staphylococcus (these two most frequently), micrococcus lanceolatus, gonococcus, bacillus coli communis, bacillus typhi abdominalis, bacillus proteus, bacillus pyocyaneus, and probably bacillus aërogenes capsulatus. (*a*) There is usually a focus of infection such as a septic wound, osteomyelitis, gonorrhœa, otitis media, empyema, a suppurating appendix, etc. (*b*) At the original point of infection the bacteria induce coagulation-necrosis with

formation of purulent thrombi which break up into septic emboli. These are carried to distant foci, where they set up embolic or metastatic abscesses. (*c*) The organs most frequently affected are the lungs, brain, spleen, liver, kidneys, and heart. Any organ or tissue in the body may be the seat of these abscesses. (*d*) Endocarditis is very liable to occur in all forms of pyæmia. (*e*) Retinitis has been noted in thirty per cent. of cases of pyæmia. Instances have been observed in which retinal hemorrhage was the only evidence of general septic infection.

SMALLPOX.

Etiology.—(*a*) Overcrowding. (*b*) Improper food. (*c*) Season, fall or winter. (*d*) Streptococci are found in the characteristic lesions, but no pathogenic micro-organism has as yet been isolated.

Morbid Anatomy.—(*a*) The characteristic lesion of smallpox is a rash. On the skin may be seen papules, umbilicated vesicles, pustules, and crusts. A shot-like feel of the papules upon the forehead and wrist is quite characteristic. (*b*) The rash may also be found upon the mucous membranes from the mouth to the rectum, but on account of the moisture the pocks are not quite so characteristic in these situations as upon the skin. In some cases there is deep ulceration, especially in the larynx, which may be followed by necrosis of the cartilages. (*c*) Swelling of Peyer's patches is not uncommon. (*d*) In the hemorrhagic form of smallpox extravasations of blood are found on the serous and mucous surfaces, in the parenchyma of the organs, in the connective tissue, and about the nerve-sheaths. They have also been observed in the bone-marrow and in the muscles. (*e*) As a rule, the spleen is markedly enlarged, but it may be small, very dark, and firm. (*f*) The heart is flabby and pale. The myocardium shows cloudy swelling and fatty degeneration. It is often dark brown in color and may be firm to the touch. The cavities contain little or no clotted blood, and the arterial trunks are nearly empty. (*g*) Lesions of the kidney are not common. It may show cloudy swelling and areas of focal necrosis, or the pelvis may be blocked with dark clots which sometimes extend into the ureters. (*h*) Absence of the scar resulting from vaccination is very often noted. (*i*) The epidermis of the hands and feet may be shed entire. The skin is sometimes plum-colored. (*j*) The face may be swollen. Hemorrhages not infrequently occur in the conjunctiva, and the cornea may be sunken.

Complications.—(*a*) Bronchopneumonia is almost invariably present in fatal cases; lobar pneumonia and pleurisy less commonly. (*b*) Albuminuria is frequent, but true nephritis is rare. (*c*) Puru-

lent changes in the arteries, bones, conjunctiva, and middle ear are common. (*d*) Ulcerative laryngitis with œdema sometimes cause death. (*e*) Myocarditis, endocarditis, and pericarditis are comparatively common. At a post mortem made by me the odor was so characteristic that, like the elder Mitchell, I think I could recognize a case of smallpox by this means alone. The physician should always vaccinate himself before making a post mortem on a smallpox case.

SPLEEN, DISEASES OF.

Amyloid Degeneration of.

Etiology.—(*a*) Prolonged suppuration (tuberculosis or syphilis). (*b*) Chronic visceral diseases. (*c*) Cachexias. (*d*) Grave anæmia. *Classification.*—(*a*) Localized. (*b*) Diffuse.

Morbid Anatomy.—(*a*) In the localized form the spleen is usually somewhat increased in size and firm in consistency, and the color may be lighter or darker than normally. On section there are seen on the surface small rice-like bodies, more or less symmetrically arranged and especially numerous about the Malpighian bodies. Care should be taken not to mistake these bodies for miliary tubercles. (*b*) In diffuse amyloid degeneration the spleen is often enormously enlarged. The outline is rounded, the organ firm, often hard in consistency, pale in color, and lardaceous or bacon-like in appearance. Both the local and diffuse varieties give the characteristic mahogany-brown color with Lugol's solution. (*c*) All the infectious granulomata of syphilis may produce marked cirrhosis of the spleen with great increase in the thickness of the capsule. At times the capsule appears as though melted tallow had been poured over its surface and allowed to dry. From the inner side of the capsule large trabeculæ extend into the splenic substance. The blood-vessels show arteriosclerotic changes. (*d*) Gummata are rare, but when present they frequently form large stellate scars.

Hyperæmia of.

Classification.—(*a*) Acute. (*b*) Chronic or passive.

Morbid Anatomy.—(*a*) Acute hyperæmia of the spleen resembles acute splenic tumor. (*b*) In chronic congestion the spleen is somewhat enlarged, firm in consistency, and the capsule and trabeculæ are thickened. The splenic pulp is dark in color, firm, and shows increase of connective tissue. The term cyanotic induration best describes the condition.

Infarcts of.

Etiology.—(*a*) Cardiac emboli. (*b*) Septic abortions. (*c*) Operations involving the rectum or the genito-urinary tract. (*d*)

Septic inflammations of the digestive tract. *Classification.*—(*a*) Mechanical or non-septic. (*b*) Septic.

Morbid Anatomy.—(*a*) Mechanical embolism of the spleen is most frequently of cardiac origin. It usually results in the formation of an anæmic infarct. This infarct is pale yellow, devoid of blood, and wedge-shaped. In the hemorrhagic form the affected area is red, quite dense, pigmented, and raised above the surrounding surface. Scars in the spleen often result from the healing of these areas of necrosis. (*b*) Septic embolism is exceedingly common. It is frequently peripheral, often pyramidal in shape, and terminates in abscess formation. (See Purulent Splenitis.)

Inflammation of.

Etiology.—(*a*) Acute infectious fevers. (*b*) Micro-organismal infection other than that of the infectious fevers. (*c*) Extension from neighboring organs. *Classification.*—I. *Acute.*—(*a*) Simple. (*b*) Purulent. II. *Chronic.*

Morbid Anatomy.—I. 1. Simple acute infection of the spleen (acute splenic tumor) (*a*) is characterized by marked increase in the size of the organ. The capsule is stretched and tense, and small hemorrhages under it are common. The color is darker than normal. (*b*) On section the contents bulge out. The Malpighian bodies are prominent in most cases, giving to the organ a marble-like appearance. When less prominent, they look like miliary tubercles, but differ from them in that they are flat, whereas miliary tubercles are not cut by the knife, and hence are elevated from the surface. In some cases they may be obliterated. The splenic pulp is dark red or brownish in color, quite soft in consistency, granular in appearance, and in the centre may be diffluent. The central mass often resembles a currant jelly clot of dark color. 2. (*a*) Purulent splenitis occurs most frequently in pyæmia and is usually the result of septic embolism. The abscesses are small, non-encapsulated, and most numerous about the cortex. The spleen is enlarged, deeply congested, soft, and friable. (*b*) A large single abscess may arise from extension and involve the greater portion of the organ. II. (*a*) Chronic splenitis may follow repeated acute attacks. It is probably most common as the result of malaria (see Malaria, page 264), syphilis, and slowly acting poisons. (*b*) The spleen is usually increased in size, firm in consistency, and on section shows marked increase of connective tissue, especially about the trabeculæ. (*c*) Its color is chocolate brown or slaty gray. The blood-vessels, especially towards the hilum, are thickened and prominent. (*d*) Perisplenitis often accompanies chronic splenitis. It may occur inde-

pendently, as in tubercular affections of the peritoneum (see Peritonitis).

Leprosy of.

In leprosy, involvement of the spleen is characterized microscopically by the presence of large ovoid mononuclear cells, usually vacuolated and containing numerous lepra bacilli. Actinomycosis of the spleen is rare, and leads to purulent inflammations.

Tuberculosis of.

Classification.—(*a*) Acute miliary. (*b*) Chronic or caseous. (*c*) Perisplenitic.

Morbid Anatomy.—(*a*) In acute miliary tuberculosis the spleen is increased in size, firm in consistency, deeply congested, and on section presents a greater or less number of symmetrically distributed, small, grayish, semi-translucent bodies, slightly raised above the surface and firmly adherent. This condition is seldom primary, but is common as a secondary lesion. (*b*) Caseous tuberculosis is attended by a variable number of irregularly distributed cheesy nodules or diffuse areas of caseation. These may be as small as a pea or as large or larger than a walnut. They not infrequently break down and form abscesses through pyogenic infection. (*c*) Tubercular peritonitis frequently occurs as an extension from peritonitis.

SPRUE (PSILOSIS).

Definition.—A chronic remittent inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in persons residing, or who have resided, in tropical or subtropical climates.

Etiology.—Apparently nothing is known of its origin.

Morbid Anatomy.—At post mortem the thoracic organs, the abdominal viscera, and the tissues generally are found to be much wasted, giving the body a mummified appearance. The bowel is exceedingly thin, and on opening it a thick layer of dirty viscid gray, tenacious mucus is seen. On removing this, areas of congestion, ulceration, pigmentation, or thickening may be found. The mesenteric glands are generally enlarged, dark, and fibrinous.

STOMACH, DISEASES OF.

Cancer of.

Etiology.—(*a*) *Heredity.*—In eight per cent. of Haeberlin's cases a history was obtained of its having existed in others of the family.

(*b*) It is more common in the male sex. (*c*) *Age.*—In Brinton's cases the age averaged fifty years; it is most common between the ages of forty and seventy. (*d*) *Ulcer.*—According to Hauser, proliferation at the edge of healing ulcers is sometimes the cause of cancer. (*e*) *Geographical Distribution.*—It is rare in Egypt and common in Switzerland. *Classification.*—(*a*) Cylindrical-celled at the pylorus. Squamous epithelioma at the cardiac end. (*b*) May be hard (scirrhus), soft (medullary), or colloid.

Morbid Anatomy.—(*a*) *Scirrhus.*—A small nodular growth occurring most often at the pylorus, often sharply defined and very hard. It is whitish on section, and no cancer juice exudes from the cut surface. Stricture of the pylorus with hypertrophy and dilatation of the stomach are common. Connective tissue is very abundant and cancer-cells few. Ulceration occurs late in the disease. (*b*) *Medullary.*—This tumor tends to become larger than the previous one. It contains much less connective tissue and is therefore softer. It involves all the coats and is not circumscribed. It ulcerates very early, and hemorrhages are frequent. As in the previous instance, metastasis is very common. (*c*) *Colloid.*—This variety usually consists of gelatinous cancer-cells in a condition of colloid degeneration. It extends over the entire stomach and metastasis is very rapid. Metastasis in all the forms affects the various tissues and organs in the following order: lymphatic glands, liver, peritoneum, omentum and intestine, pancreas, pleura, lung, and spleen. The squamous variety is a somewhat flat tubular swelling involving the superficial layers. It may constrict the œsophageal orifice and cause atrophy of the stomach.

Gastrectasis.

Definition.—Dilatation of the stomach.

Etiology.—I. *Pyloric Stenosis.*—(*a*) Carcinoma. (*b*) Congenital. (*c*) Hypertrophy of the pyloric sphincter. (*d*) Cicatrix of an ulcer. (*e*) Peritoneal adhesions. (*f*) Cancer of the head of the pancreas or other structure pressing on the duodenum. (*g*) Spasm of the sphincter. II. *Atony of the Gastric Walls.*—(*a*) From chronic gastritis. (*b*) Excessive ingestion of solids and liquids. (*c*) Traumatism. (*d*) Surgical intervention. (*e*) Serious infectious diseases. (*f*) Neurasthenia (Hemmeter). *Classification.*—(*a*) Acute (rarely seen). (*b*) Chronic.

Morbid Anatomy.—At first there is hypertrophy of the muscular walls. Soon, however, interstitial sclerosis comes on, the stomach may become either pyriform or hour-glass in shape, and the mucous coat is thrown into exaggerated folds. As atrophy advances all the layers of

the stomach become thinner; the bundles of muscles are separated by connective tissue; the surface may show evidences of pigmentation and petechial hemorrhage; and while the serous surface sometimes remains unaltered, it is usually thick, pale, and opaque.

Gastritis.

Definition.—Inflammation of the stomach.

Etiology.—I. *Acute.*—(*a*) Errors in diet both as regards quantity and quality. (*b*) Irritant poisons. (*c*) Mechanical: external (severe injury to the epigastrium); local (fish-bone, etc.). (*d*) Thermal (hot or cold ingesta). (*e*) Infectious diseases. (*f*) Psychic shock (grief, sorrow, etc.). (*g*) Extension of inflammation. II. *Chronic.*—(*a*) Follows repeated acute attacks. (*b*) Slow poisons (alcohol, tobacco, gout, rheumatism). (*c*) Diet. (*d*) Anæmia and chlorosis. *Classification.*—I. *Einhorn* (*Acute Gastritis*).—(*a*) Simple. (*b*) Phlegmonous. (*c*) Toxic. II. *Chronic Gastritis.*

Morbid Anatomy.—I. (*a*) In this condition the mucous membrane is hyperæmic, swollen, and covered with profuse creamy mucus. There are localized areas of ecchymosis and often small erosions. In severe cases there is considerable denudation of epithelium, with perhaps an exudate of grumous blood. (*b*) Phlegmonous or suppurative gastritis may exist in two forms: the abscesses may be small, multiple, and miliary, or they may be diffuse. The pyloric end is most commonly involved. The submucous layer is most extensively altered. It is swollen, œdematous, purulent, and sometimes even bloody. The mucous membrane overlying the abscess may be normal in appearance, it may slough off, or, again, it may be swollen and hemorrhagic. On the other hand, the surface is sometimes studded with numerous areas of focal necrosis of a yellowish appearance, and, on section, may discharge pus. Diphtheritic gastritis sometimes follows laryngeal or pharyngeal diphtheria, and frequently accompanies pyæmia, scarlet fever, variola, and malignant endocarditis. In this form of gastritis we find a variable number of circumscribed areas of false membrane firmly adherent to the underlying structures and leaving a raw surface when removed. It is apt to attack particularly the crests of the rugæ. The diphtheritic patches are usually surrounded by areas of more or less pronounced congestion. (*c*) In toxic gastritis the appearance of the viscus depends upon the amount of contained food at the time of ingestion and the concentration and kind of poison. If the latter is diluted, the mucous membrane alone suffers; if concentrated, all the coats may be involved. Alkalies appear to be more destructive than acids, the lesions produced resembling those of an intense congestion, more or less localized. In

very severe cases perforation may follow. Sloughs or ulcers are almost invariably found where the poison has been concentrated. Mycotic gastritis may be due to: (*a*) Anthrax. (*b*) Favus. (*c*) Thrush. II. *Chronic.*—(*a*) *Hypertrophic.*—This may be localized or diffuse. In the former case there can be seen numerous mucous polyps over the affected area. These warty elevations show considerable cystic degeneration. In the diffuse variety the stomach is almost invariably enlarged and the walls thickened, particularly the mucous coat, which is decidedly velvety both to sight and touch. Besides being swollen, rugæ are often present in exaggerated folds. Petechial hemorrhages and areas of pigmentation are common. There are often evidences of previous ulcerations (cicatrices). The stomach frequently contains a variable quantity of thick, tenacious, sour-smelling, greenish mucus. (*b*) *Atrophic.*—When this variety of the disease exists the walls of the stomach become thinner. There is connective tissue overgrowth, which by its contraction causes the epithelial cells to undergo degeneration and disappear. The mucous membrane is thin, smooth, and pigmented.

SUNSTROKE (THERMIC FEVER, INSOLATION).

Morbid Anatomy.—(*a*) There are no characteristic changes. Rigor mortis comes on early. Lividity and putrefactive changes develop rapidly after and even before death. (*b*) Venous engorgement is extreme, particularly in the cerebrum. (*c*) The left ventricle of the heart is contracted; the right is dilated and may be full of blood imperfectly coagulated and deficient in oxygen. (*d*) The blood is fluid, dark in color, and acid in reaction. (*e*) Parenchymatous changes occur in the liver and kidneys. (*f*) Petechial patches may appear in the subcutaneous and subserous tissues. The elevation of temperature is often remarkable, and it is extremely disagreeable to make an autopsy in these cases, as I have done, soon after death, with a temperature of 106° F. In a case of mine of stramonium poisoning, with a temperature of nearly 110° F., the clinician had diagnosed sunstroke.

SYPHILIS.

Etiology.—Lustgarten and others have described specific organisms, one of which has been definitely accepted. *Classification.*—I. *Acquired Form.*—(*a*) Primary. (*b*) Secondary. (*c*) Intermediate period. (*d*) Tertiary. II. *Hereditary Form.*—(*a*) Primary. (*b*) Secondary.

Morbid Anatomy.—The following lesions should be looked for in making a post mortem: (*a*) The initial lesion or its scar. (*b*) Lym-

phatic enlargement, especially of the groin, neck, and elbow. (*c*) Various skin lesions and thinness of the hair. (*d*) Mucous patches. (*e*) Onychia and dactylitis. (*f*) Gumma in the viscera, skin, subcutaneous tissues, muscles, etc. (*g*) Parotitis. (*h*) The bones for periostitis or osteomyelitis. (*i*) The eye for iritis or choroiditis. (*j*) The bowels for stricture, especially the rectum. (*k*) The nervous system for tabes, dementia paralytica, and other forms of sclerosis. I. The lesions found in the *primary stage* are: (*a*) The chancre. This begins as a small red papule, usually situated at the junction of the skin and mucous membrane. It gradually enlarges and breaks in the centre, leaving a small ulcer with indurated edges and base. (*b*) The neighboring lymphatic glands are enlarged and hard. II. *Secondary Stage.*—(*a*) Cutaneous eruptions of all forms. As a rule, the syphilide is polymorphous, varying in form from an erythema to a pustular eruption. It is symmetrically distributed and of a reddish-brown or copper color. It appears most frequently on the chest, abdomen, and flexor surfaces of the arms. (*b*) The mucous patch is a softened and macerated epithelium, and appears on the mucous membrane or on the moist regions of the skin. It is most frequently found in the mouth, in the throat, and about the anus. The mucous patch is irregularly shaped, non-inflammatory, and does not discharge pus. (*c*) The hair of the scalp is decidedly thin. (*d*) Ulcers may be seen on the tonsils and larynx. (*e*) There may be warts about the vulva and anus. (*f*) Iritis is common; retinitis rare. (*g*) The finger-nails may be diseased, forming dry or moist onychia. (*h*) Periostitis may be present. III. In the *intermediate stage* there are but few lesions: (*a*) gumma of the testicles, and (*b*) chorioiditis are the only ones found. IV.—*Tertiary Stage.*—(*a*) The late syphilides show a tendency to ulcerate and destroy the deeper layers of the skin, leaving scars. Rupia may develop. (*b*) The gummata are the characteristic lesions, and may be hard or soft. The former develop in the internal organs and in the mucous membranes. They most frequently terminate in cicatrization, forming stellate scars which often cause marked deformities. Soft gummata are found in bones, skin, etc. They tend to break down and ulcerate, leaving chronic indolent, often serpiginous, sores. (*c*) When there has been prolonged suppuration, amyloid degeneration of the liver, spleen, and kidneys often occurs. This is especially true with regard to rectal syphilis in women. (*d*) *Circulatory System.*—The heart frequently shows sclerotic changes of the valves, especially about the aorta. (*e*) The blood-vessels present arteriosclerosis or atheromatous changes. (*f*) In the central nervous system scleroses of the brain and cord and gumma are common. V. *Congenital Syphilis.*—

(*a*) At birth the infant is usually apparently healthy, but it may present well-marked lesions. (*b*) There is wasting, and pemphigus is noticed on the hands and feet. (*c*) The lips may be ulcerated and the mouth and anus fissured. (*d*) There is inflammation of the nasal mucous membrane; hyperæmia with papillary infiltration is present and necrosis of the bone may occur. (*e*) The spleen and liver are enlarged. (*f*) The lungs may present the lesions of white pneumonia or miliary gummata. (*g*) The long bones usually show characteristic changes, and the epiphysis may be separated. (*h*) Later the child looks prematurely old. The teeth are wedge-shaped and the cutting edges notched (Hutchinson's teeth). (*i*) Eye lesions may be seen as interstitial keratitis. (*j*) Dactylitis is not uncommon.

Syphilis of the Brain and Cord.

Morbid Anatomy.—(*a*) Gummata are usually multiple, varying in size from a pea to a walnut. In the cerebrum they occur along the sulci. Heubner describes two forms. In the first variety they are grayish or grayish red in color, soft, and not sharply defined. On section they are moist and exude a small amount of juice. In the second form they are quite hard and dry. Their outline is distinct. On section they may be cheesy and look not unlike tubercular growths. (*b*) Gummatous arteritis and sclerosis of both arteries and nerve tissue may exist. (*c*) There may be softening due to obstruction of the blood-vessels. Recently in Philadelphia a man was condemned to death for killing a person in cold blood. A commission of experts pronounced him sane. The man committed suicide by hanging, and I found at the post mortem numerous gummata of the brain, situated especially in the right temporal and frontal regions.

Syphilis of the Circulatory System.

Morbid Anatomy.—(*a*) Gummata are rare. (*b*) Fibrosis of the heart-muscle is common. (*c*) Sclerosis of the valves is frequent. (*d*) Arteriosclerosis, aneurism, and endarteritis obliterans are common.

Syphilis of the Gastro-Intestinal Tract.

Morbid Anatomy.—(*a*) The œsophagus is rarely affected. Ulceration or stenosis may be present. (*b*) Ulcers, phlegmonous inflammations, or abscesses may be found in the pharynx. (*c*) Ulcers may occur in the small intestine and cæcum. (*d*) The rectum is not infrequently the seat of cicatricial contraction. This lesion is most frequently seen in women.

Classification.—The lesions that syphilis produces in the gastro-

intestinal tract are (*a*) chancre, (*b*) ulcers, (*c*) localized fibrous patches, (*d*) gummata, (*e*) miliary nodules, (*f*) condylomatous masses.

Syphilis of the Kidneys.

Morbid Anatomy.—(*a*) Gummata are not infrequent. (*b*) Acute syphilitic nephritis may occur. (*c*) Chronic interstitial nephritis is more common.

Syphilis of the Liver.

Morbid Anatomy.—(*a*) In diffuse syphilitic hepatitis there is marked fibrous change. The organ is hard, firm, and resistant. The disease usually begins with a perihepititis, which frequently causes adhesions to the surrounding structures. With contraction of the fibrous tissue great deformities of the liver become manifest. Capillary bile-ducts may be present in abundance in the cirrhosed portion. (*b*) The smaller gummata are pale-grayish nodules, the larger ones pale yellowish in color. Usually they are multiple. Although they may be present in any part of the organ, the most common situation is at the junction of the right and left lobes. Great deformity results from healing and contraction.

Syphilis of the Lung.

Morbid Anatomy.—(*a*) In white pneumonia of the fœtus the affected lung is heavy and airless. On section it presents a grayish-white appearance. (*b*) Hereditary gummata are small in size, grayish in color, firm in consistence, and more or less symmetrically distributed throughout the lung. (*c*) Acquired gummata vary in size from a pea to a goose's egg. They are grayish yellow in color and are embedded in connective tissue. The parts around them are hard and brawny and of a glossy lustre. (*d*) There may be a fibrous interstitial pneumonia in which the lesions are hard, large, and pale or dark grayish red in color. The middle of the right lung or either apex is the part most frequently involved. (*e*) The pleura is thickened. (*f*) Endocarditis may extend to the hepatic artery and portal vein.

Syphilis of the Testes.

Morbid Anatomy.—(*a*) Gummatous growths usually involve the epididymis, which becomes a hard mass, from the size of a bean to that of a walnut. It affects the head more commonly than the body of the epididymis. (*b*) In interstitial orchitis the progress of the disease is slow. The organ is larger than normal and distinctly harder to the touch. The overlying skin is not adherent and there is no tendency to suppuration.

SYRINGOMYELIA.

Definition.—Syringomyelia is a chronic affection of the spinal cord characterized anatomically by the pathological formation of cavities in its gray matter, and clinically by peculiar disturbances of sensibility associated with trophic disorders.

Etiology.—(*a*) Embryological malformations. (*b*) A gliosis. (*c*) Traumatism. (*d*) Development of embryonal neurogliar tissue in which hemorrhage or degeneration takes place with the formation of cavities.

Morbid Anatomy.—(*a*) The characteristic lesion is a cavity which forms in the cord in or near the central canal and extends into the gray matter of the anterior, or more frequently the posterior horns. It is most often situated in the cervical and thoracic portions of the cord. (*b*) On transverse section the cavity may be oval, circular, or narrow and fissure-like, or it may present the appearance of two or more cavities independent of each other or intercommunicating. (*c*) The contents of the cavity are usually a colorless liquid. Occasionally it may be a yellow or brown gelatinous substance, or it may consist of blood and the products of its degeneration. The white matter of the cord in moderate cases is unaffected, but where the cavity is large and pressure from the sclerotic tissue has become great, the white matter is in its turn involved, being crowded to the periphery and more or less unable to carry on its functions.

TENDONS, SHEATHS, AND BURSÆ, LESIONS OF.

Tendons and Sheaths.

Morbid Anatomy.—(*a*) Acute tenosynovitis may be either simple or purulent. It is usually the result of wounds or bruises. (*b*) The purulent form is caused by pyogenic infection. The pus shows a marked tendency to burrow between the sheath and the tendons, sometimes for considerable distances. (*c*) In the dry form deposits of fibrin are found upon the inner surfaces of the sheath, and may give rise to a rubbing or creaking sensation. (*d*) Chronic tenosynovitis is generally gouty or rheumatic or the result of the healing of wounds. Calcareous deposits are particularly common in this form. (*e*) Tuberculous tenosynovitis may be primary or secondary. It may be miliary or caseous. (*f*) Hygroma is the result of a chronic irritation causing an increased secretion of serous fluid beneath the sheath of the tendon. It forms a cyst-like mass which frequently contains rice-like bodies. (*g*) Arborescent lipoma is a rare condition. It is a papillomatous

outgrowth consisting mainly of fat, growing from the synovial lining. These cases often come to the physician to be treated for rheumatism.

BURSÆ.

MORBID ANATOMY.—(*a*) Acute bursitis, due usually to injury, may be serous, serofibrinous, or purulent. (*b*) Chronic bursitis occurs generally in the form of a hygroma. Housemaid's knee is the most common form. Loose bodies are frequently found in these cysts. (*c*) Tuberculosis may be primary or secondary, miliary or caseous. (*d*) A ganglion is a round oval or lobulate cyst, varying in size from a pea to a pigeon's egg, and containing a reddish-yellow crystalline jelly or colloid material. It is probably the result of the recurrence of slight injury.

TETANUS.

ETIOLOGY.—The bacillus of tetanus is a slender rod usually growing in long threads. It is motile, grows on ordinary media at ordinary temperatures, and is anaërobic. It stains readily, but does not retain the stain very well.

MORBID ANATOMY.—(*a*) The bacilli develop at the site of the wound, which is usually of a penetrating character, and do not invade the blood or organs, except very rarely late in the course of the disease. (*b*) No characteristic lesions have been found. (*c*) The condition of the wound depends upon the kind and extent of the injury. (*d*) The central nervous system shows congestion, with perivascular exudations and granular change in the nerve-cells. Some investigators have found swelling and areas of disintegration in the gray matter of the cord, with exudation of a finely granular material and disintegrated blood. (*e*) In tetanous neonatorum the umbilicus may be inflamed. (*f*) The rectus muscle has been found ruptured as the result of a spasm. (*g*) Death may occur from heart-failure or asphyxia.

THRUSH.

ETIOLOGY.—The *Oïdium albicans,* or thrush fungus.

MORBID ANATOMY.—(*a*) The mouth, tongue, cheeks, etc., are more or less densely covered with minute, slightly raised, white spots, which are quite firm and adherent to the mucous membrane. When scraped off and examined microscopically, the characteristic fungus is seen. (*b*) Occasionally the fungus invades the œsophagus and grows to such an extent as seriously to obstruct its lumen.

TUBERCULOSIS.

Definition.—Any morbid lesion produced by or through the agency of the tubercle-bacilli.

Etiology.—The tubercle-bacillus is a rod-shaped micro-organism, measuring in length about one-half the diameter of a red corpuscle and in width two-tenths of a micron. It is bent upon itself, grows best on agar containing glycerin, stains with difficulty, but retains the stain tenaciously. The best method of staining is by carbol-fuchsin and Gabbett's solution. When stained it often has a beaded appearance. It is morphologically similar to the bacillus of leprosy and the smegma-bacillus.

Classification of Tuberculous Lesions.—I. *Acute.*—(*a*) Miliary tuberculosis. (*b*) Caseous pneumonia or phthisis florida. (*c*) Tubercular ulcerations. II. *Chronic.*—(*a*) Diffuse tuberculosis, ulcerative phthisis, or caseous tuberculosis. (*b*) Fibroid phthisis. (*c*) Cold abscesses. III. *Modes of Invasion.*—(*a*) Aërogenous. (*b*) Lymphogenous. (*c*) Hæmatogenous. IV. *Characteristic Lesions of Tuberculosis.*—(*a*) Miliary tubercle. (*b*) Caseation. (*c*) Cold abscesses. (*d*) Ulceration. *Characteristics of Tuberculous Lesions.*—(*a*) Miliary tubercle is a small nodule about the size of a millet-seed, grayish white in color, semi-translucent, raised above the surface, and primarily adherent to the surrounding structures. Microscopically the centre of a typical tubercle shows one or more giant cells, the centres of which are necrosed, the nuclei being situated at the periphery or at the poles. These are surrounded by proliferated cells which are ovoid, cuboidal, or fibrillated, with somewhat pale vesicular nuclei (epithelioid cells); around these again is an area composed mainly of leucocytes, free nuclei, sometimes giant cells, and a small amount of fibrin. (*b*) In caseation or diffuse tuberculosis two or more miliary tubercles agglutinate, isolating the intervening healthy tissue and cutting off its blood-supply. The necrosed area loses symmetry of shape and arrangement and undergoes fatty degeneration. The area is yellowish in color, soft or firm in consistence, and is surrounded by an inflammatory zone. Microscopically the cells composing the tubercles fail to stain, except occasionally here and there a nucleus. There is an infiltration of leucocytes, especially towards the periphery, where proliferated cells and sometimes giant cells may also be found. There is an almost complete absence of blood-vessels. (*c*) Cold abscess is most frequently found in association with tuberculosis of the vertebræ. It is frequently seen as a "psoas" abscess. The capsule of this abscess is more or less imperfect. It does not present the ordinary charac-

teristics of a pyogenic membrane, the limiting wall being composed mainly of broken-down tubercular tissue with more or less perfectly formed tubercles. The contents of the abscess are pale and of a somewhat watery consistence, composed mainly of broken-down cells, fatty *débris,* and water. Bacteriologically the contents of the abscess are usually sterile. (*d*) Tubercular ulcerations are perhaps best seen in the intestine, where they may appear in the large or in the small bowel. They also occur in the nose, larynx, and pharynx. The tuberculous ulcer is usually irregular in outline; the edges are rough, terraced, and not undermined. The floor of the ulcer is caseous or studded with miliary tubercles. In the bowel it does not evince any tendency to perforate. In the nose, larynx, and pharynx it is apt to spread superficially. V. *Distribution of Tubercles in the Body.*—(*a*) The lungs are most commonly affected. In two hundred and seventy-five cases out of a thousand autopsies, the lungs were, with two or three exceptions, involved in all. Other organs were affected as follows: (*b*) Intestines in sixty-five cases, (*c*) peritoneum in thirty-six, (*d*) kidneys in thirty-two, (*e*) brain in thirty-one, (*f*) spleen in twenty-three, (*g*) generative organs in twenty, (*h*) liver in twelve, (*i*) pericardium in seven, and (*j*) heart in four. (Osler.) VI. *Fate of Tuberculous Lesions.*—Tubercular lesions may terminate—(*a*) In resolution, which is rare. (*b*) In fibroid changes. (*c*) In caseation or suppuration. (*d*) In calcification. (*a*) Resolution sometimes take place when the area of tuberculosis is small, the blood-supply good, and the patient under favorable conditions, especially when leading an out-door existence. (*b*) In healing by fibroid change the area affected is first encapsulated and then by gradual pressure and absorption the affected area is removed, leaving a scar. (*c*) Caseation is by far the most common result of all tubercular lesions. The process has been already described. Suppuration in tubercular lesions is the result of the introduction of pyogenic organisms. (*d*) Calcification is the most fortunate ending of the tuberculous process, and it is estimated by careful observers that seventy-five per cent. of all persons who die after the age of forty years show this form of tuberculosis in their lungs or pulmonary glands.

Tuberculosis of the Alimentary Tract.

Etiology.—This form may be (*a*) Primary in the mucous membranes. (*b*) Secondary to the disease of the lungs. (*c*) It occurs rarely through extension from the peritoneum.

Morbid Anatomy.—I. *Mouth.*—(*a*) Primary tuberculosis, which is usually miliary. The tonsils are more often affected primarily than

was formerly supposed. (*b*) Secondary to tuberculosis of the face, larynx, or lung. It may attack the tongue or cheeks and be miliary or caseous. II. *Œsophagus.*—(*a*) Primary tuberculosis is very rare. (*b*) Secondary tuberculosis through extension from the lungs or larynx is comparatively common. (*c*) The lesions may be miliary, caseous, or ulcerative. III. *Stomach.*—(*a*) Tuberculosis of the stomach is comparatively rare. It is apt to occur in the form of miliary tuberculosis of the serous membrane or of the muscularis. IV. *Intestines.*—The lesions generally occur in the ileum, cæcum, colon, and rectum. The most frequent seat is in the ileum, just above the ileocæcal valve, as it is here that stasis of the intestinal contents occurs and a favorable opportunity is given for the growth of the tubercle-bacillus. (*a*) The most common lesion is the ulcer, which is usually ovoid in shape; it may be round or transverse to the long axis of the bowel. It is usually situated near the attachment of the mesentery. (*b*) The submucosa and muscularis are usually involved, and colonies of young tubercles may be scattered over the serous membrane. (*c*) There may be solitary or multiple areas of cicatricial tissue. (*d*) *Fistula in ano* is quite common. V. (*a*) The liver is constantly involved in general tuberculosis. It is pale in color, often fatty, and presenting miliary tubercles or caseous masses which may break down into numerous small abscesses, especially about the smaller bile-ducts. (*b*) There may be a slight increase in the connective tissues, leading to tubercular cirrhosis.

Tuberculosis of the Brain and Cord.

Classification.—(*a*) Acute miliary infection. (*b*) Chronic meningo-encephalitis. (*c*) Solitary tubercles.

Morbid Anatomy.—I. *Acute Miliary Tuberculosis.*—(*a*) This is usually secondary to tuberculosis of the lungs, bronchial glands, or bones. (*b*) Miliary tubercles occur most frequently in the pia and arachnoid of the cerebellum, next in the cerebrum, then in the pons. They follow the direction of the blood-vessels. They are apt to lead to obliteration of the vessels and thus cause softening and necrotic changes. Serous, seropurulent, or serofibrinous exudate is also present. (*c*) This acute process may result in acute inflammation of the meninges, principally the pia and arachnoid. It is spoken of usually as acute hydrocephalus. This is most pronounced towards the base of the brain and occurs most frequently in children. I have found tubercle-bacilli in fluid removed by Quincke's lumbar puncture. II. *Chronic Meningo-Encephalitis.*—The membranes at the base of the brain are most often involved, next in frequency the optic chiasm, the Sylvian fissure, and the interpeduncular space. The membranes are thickened,

firmly adherent, and covered with a fibrinous, purulent exudate. The convolutions are flattened and the sulci obliterated. The cerebral substance is more or less œdematous. The lateral ventricles are dilated and contain a turbid fluid. III. *Tuberculous Tumors of the Brain.*—(*a*) Solitary tubercles are found most usually about the cerebellum. As a rule, they are attached to the meninges, often to the pia mater. (*b*) Cerebral softening from pressure is not uncommon. The tubercles vary in size from a pea to a small orange. They are grayish yellow in color, caseous, and usually firm and hard, but the centre may be semi-fluid. They are, as a rule, surrounded by a soft translucent tissue. (*c*) They may calcify.

Tuberculosis of the Circulatory System.

Morbid Anatomy.—(*a*) Primary tuberculosis of the larger vessels is unknown; secondary lesions are not infrequently found if carefully searched for. (*b*) In the lungs, brain, and other organs the smaller arteries are usually involved in acute infiltration which leads to thrombosis. (*c*) Tubercles may develop in the walls of the vessels and undergo softening, which may result in hemorrhage or a wide-spread distribution of the tubercular infection.

Tuberculosis of the Genito-Urinary System.

Etiology.—(*a*) Most common in males. (*b*) Age from twenty to forty years.

Morbid Anatomy.—I. *The Kidneys.*—(*a*) These organs are frequently the seat of an acute miliary infection, which may be primary or secondary. The disease begins most frequently in the pyramids and calyces. It may be limited to the areas supplied by a single blood-vessel. (*b*) Necrosis and caseation rapidly follow. One or both organs may be affected, but at autopsy both are found to be enlarged. (*c*) Not infrequently one kidney may be completely destroyed and converted into a series of cysts; these contain a cheesy substance and lime salts may be deposited in their walls. (*d*) The walls of the pelvis may be thickened and cheesy, and the mucous membrane converted into a necrotic ulcerating mass. The ureters are usually thickened, caseous, or ulcerated. II. *The Bladder.*—(*a*) Infection of this organ is nearly always secondary to infection elsewhere, particularly in the pelvis of the kidney. The bladder is small, shrunken, thickened, and surrounded by sclerosed tissue. The mucous membrane is red, irregular, and fungoid in appearance. Minute gray tubercles may be seen. In advanced cases ulcers are found. (*b*) To find tubercle-bacilli in the urine centrifugation should be employed, and the precipitate stained in the usual man-

ner for showing these organisms. Care must be taken not to get the smegma-bacillus; it is, therefore, advisable that the urine be collected with the strictest precautions. III. *The Testes.*—Infection may occur before the second year. It may be secondary to peritoneal tuberculosis. At times the greater part of the testis is destroyed, its stroma being replaced by a softened or still firm caseous deposit, which may be softened in the centre. IV. Tuberculosis of the *ureters* is very rare. V. *Salpingitis.*—The oviducts are enlarged, the walls thickened and infiltrated, and the contents cheesy. It is usually bilateral.

Tuberculosis of the Larynx.

Morbid Anatomy.—The lesions may be primary or secondary, usually the latter. The lesions found are—(*a*) Miliary tuberculosis. (*b*) Diffuse tuberculosis. (*c*) Ulceration.

Tuberculosis of the Lung.

Classification.—I. *Acute.*—(*a*) Miliary tuberculosis. (*b*) Phthisis florida, showing itself as bronchopneumonic tubercles, as lobar-pneumonic tubercles, or as a combination of both. II. *Chronic.*—(*a*) Ulcerative phthisis. (*b*) Fibroid phthisis.

Morbid Anatomy.—I. *Acute.*—(*a*) In acute miliary tuberculosis the lesions are usually present in both lungs. They are frequently so small and transparent that they may be overlooked on macroscopic examination. At other times they are aggregated in localized spots or even become diffuse. In the latter case the lung is increased in size, is firm in consistence, in color is a darker shade of red, is heavier, and crepitates. (*b*) Phthisis florida, or acute phthisis with formation of cavities, presents a varied appearance. One lobe, or more or less of the whole lung, may be affected. The organ is heavy; the implicated portions do not collapse and are firm and airless. The pleura is covered with a thin exudate. On section the condition may resemble red or gray hepatization or an intermediate stage between them. In other instances the lung presents a mottled appearance, some areas being intensely congested, others exhibiting a characteristic pale-gray gelatinous exudate, others caseous degeneration and not infrequently cavity formation. Recently affected areas of pulmonary tissue with croupous pneumonia are often seen. II. *Chronic.*—(*a*) In ulcerative tuberculosis, apical involvement in relation to implication at the base exists in the proportion of five hundred to one, according to Kidd. There are varied lesions. First, there are caseous nodules, which are grayish, white, or yellow in color. Second, cavities may exist, which, if the case is acute, have walls made up of soft caseous masses. In

the more chronic cases these walls are replaced by pyogenic membranes of greater or less density. Frequently trabeculæ are seen in the walls; these are the blood-vessels which have resisted the tubercular process. Third, pneumonic areas and evidences of chronic bronchitis are seen. Fourth, some thickening of the pleura is constant. This may be merely an acutely inflamed area rubbing against a corresponding area on the parietal pleura or it may be tightly adherent to it. Not infrequent perforation causes a pyopneumothorax. Fifth, enlarged bronchial glands are discovered which are caseous and often pigmented. Lastly, the bronchi are thickened and the lumina of the smaller ones frequently obliterated. The larger tubes show caseous deposits in the submucous and fibrous coats. (*b*) In fibroid phthisis the organ is permeated with interstitial overgrowth. In some cases the interstitial change is most prominent; in others the tubercular process is slightly more marked. The unaffected portions of the lung are largely emphysematous and pigmentation is considerable. The right ventricle and sometimes the whole heart are hypertrophied.

Tuberculosis of the Lymphatic Glands.

Morbid Anatomy.—(*a*) Location, most frequent in the cervical chain. (*b*) Extension opposite that of the lymphatic stream. (Trevés.) I. *Chronic Form.*—(*a*) Hard. (*b*) Non-adherent. (*c*) Yellowish white in color. (*d*) Little tendency to break down and suppurate. (*e*) Tendency to be localized. (*f*) Overgrowth of connective tissue considerable. II. *Less Chronic Form.*—(*a*) Not as dense. (*b*) Tendency to become adherent. (*c*) Gray or grayish white in color. (*d*) Tendency to liquefy and suppurate. (*e*) Connective tissue less in amount. (*f*) Tubercle-bacilli more abundant.

Tuberculosis of the Mammary Gland.

Etiology.—(*a*) Female sex. (*b*) Strumous temperament. (*c*) Age from the fortieth to the sixtieth year.

Morbid Anatomy.—(*a*) Induration is at first small and very slowly increases in size. (*b*) The nipple may be retracted. (*c*) The skin over the gland becomes riddled with sinuses with indurated edges. (*d*) Associated with lymphatic enlargement, tuberculosis of bone, or other tubercular involvement near the gland.

Tuberculosis of the Peritoneum.

Classification.—I. Miliary. II. Chronic diffuse. III. Ulcerative.

Morbid Anatomy.—I. *Miliary Form.*—(*a*) On opening the ab-

dominal cavity the serous membranes seem to be covered to a greater or less extent with miliary tubercles, which are present in the mesentery and the omentum also. Frequently the gray nodules follow the distribution of the blood-vessels. (*b*) In many cases there is little or no inflammatory exudate, although petechial hemorrhages are common. (*c*) The peritoneum, however, has not its normal shining surface, but is usually pale, somewhat sticky, and lustreless. (*d*) In many cases there is an effusion of straw-colored or bloody fluid which may amount to a litre or more. It contains a considerable amount of albumin and some cells. The exudate is rarely purulent. II. *Chronic Diffuse Form.*—(*a*) The abdominal viscera and peritoneum are bound together by tough, firm, membranous bands of organized exudate and the peritoneal cavity is obliterated. (*b*) The intestinal coils are shortened and contracted, while the mesenteries and omentum are enormously thickened. (*c*) The capsules of the liver and spleen undergo extreme thickening, varying from a few millimetres to several centimetres. The organs are rough and irregular in outline. III. *Ulcerative Form.*—(*a*) There is a formation of caseous masses that vary in size from a pea to a marble, and which tend to run together and break down, forming more or less extensive ulcerating surfaces. (*b*) Adhesions are formed of a serofibrinous or seropurulent character. (*c*) The new tissues are apt to become pigmented and of a gray or almost black color. (*d*) The intestinal walls are very friable. (*e*) Fistulæ, opening at various points, are not infrequent.

Tuberculosis of the Serous Membrane.

Morbid Anatomy.—There are three groups of cases: (*a*) Acute miliary tuberculosis, which may develop very rapidly and is accompanied by more or less serous but turbid exudate. (*b*) A chronic form characterized by exudation, the formation of cheesy masses, and a tendency to suppuration. (*c*) Cases in which the tubercles are hard and fibroid, the membranes much thickened, but with little or no fluid exudate. In these cases there may be no visceral tubercles.

Tuberculosis of the Skin.

Lupis Vulgaris.

Definition.—A cutaneous form of tuberculosis, characterized by the formation of nodules, which tend to break down, producing more or less ulceration.

Etiology.—The tubercle-bacillus. It is found in very few numbers.

Morbid Anatomy.—(*a*) It starts as a small nodule, reddish brown in color and of soft consistence. These nodules vary in size from a

pin-head to a cherry and quickly break down and ulcerate. The ulcers are more or less rounded and have a red base covered with granulations. The intervening tissues show diffuse infiltration and fibrous hyperplasia. Warty excrescences may develop in the epidermis or in the floor of the ulcers. The face is the most common seat of the disease. (*b*) In lupus of the larynx the lesion is surrounded by hyperæmic, œdematous tissue. In the course of time smooth, hard nodules appear, causing great deformity of the parts. Softening and ulceration give the larynx a worn-eaten appearance. The disease follows the lymphatic channels.

Warts, Anatomical.

Morbid Anatomy.—These small papillary outgrowths are frequently seen on the hands of those who make many autopsies. The process is chronic, and, as in the case of one of my helpers in the post-mortem room at Blockley, may give rise to general tuberculosis. The bacilli are few, and are best demonstrated by inoculation of some of the secretion into a guinea-pig. The animal lives for a longer period of time than is usual when it is inoculated with tuberculous material taken from other sources.

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